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THE CLINICS

OF

JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

VOLUME IV, 1915

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PHILADELPHIA AND LONDON
W. B. SAUNDERS COMPANY

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA. PRICE PER YEAR: $8.00; FOREIGN, 35 SHILLINGS. BOUND IN CLOTH, $12.00 PER YEAR

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RICHMOND STEPHENS, M. D.

Volume IV
THE CLINICS
OF
JOHN B. MURPHY
Number 1

MURPHY'S CLINICAL TALKS ON SURGICAL AND GENERAL DIAGNOSIS

INTESTINAL FISTULAS—A DIAGNOSTIC TALK FOLLOWED BY FOUR CASES WITH COMMENTS

Dr. Murphy: What is an intestinal fistula?

Intern: It is a break in the continuity of the intestinal canal.

Dr. Murphy: It is more than that; a diverticulum would answer that definition quite as well.

Intern: A fistula communicates with the outside of the body.

Dr. Murphy: Do all fistulas communicate with the outside?

Intern: They communicate at least with some canal leading to the outside.

Dr. Murphy: No. A fistula may equally well communicate with other structures. An intestinal fistula may open at one end into the intestine and at the other on the surface or into the interior of some other organ or viscus. An intestinal fistula may lead up into the pleural cavity. I have seen an intestinal perforation penetrating into the pancreas, producing a pancreatic abscess and ultimately resulting in a fistula connecting the pancreatic duct with the bowel. I have seen an intestinal fistula connecting the bowel with the interior of the liver, where it opened into a liver abscess cavity. It is not so very rare to have a subphrenic abscess form one end of an intestinal fistula. Tell us some of the different varieties of intestinal fistulas.

Intern: An artificial anus.

Dr. Murphy: An artificial anus is produced surgically. Name some of those that are produced pathologically.

Intern: A fecal fistula.

Dr. Murphy: That doesn't mean much more than an intestinal fistula.

Intern: One that communicates with the outside.

Dr. Murphy: Tell us what are the common pathologic conditions that lead to fistulas, then perhaps you will be able to deduce for us the different varieties. What is the most common type of intestinal fistula that you have seen during your internship?

INTERN: The post-operative fistula.

DR. MURPHY: That is correct. After operations for what?

INTERN: Appendicitis usually.

DR. MURPHY: Yes; but if you say after a lesion of the appendix, rather than after an appendicitis, then you also include the other lesions of the appendix, such as tuberculosis and malignancy, which are not rarely the cause of a fistula. Where are the openings most frequently located?

INTERN: At the side of the rectus muscle.

DR. MURPHY: I mean the openings in the bowel, not on the skin.

INTERN: In the cecum.

DR. MURPHY: Where?

INTERN: Along the stump of the appendix.

DR. MURPHY: Yes, near the stump of the appendix. That is the most common location. What is the next most common site—one where ulceration is the cause of the fistula?

INTERN: The appendix.

DR. MURPHY: No, not at all.

INTERN: Lymphatic tissue.

DR. MURPHY: That's a bad guess. I think I'll have to tell you. I refer to the ulcerations of the cecal wall not directly adjoining the appendix stump. An abscess of appendiceal origin spreads outside and around the cecum and the pus erodes and necrotizes the wall of the cecum, forming an ulcer on the peritoneal surface, which finally penetrates into the lumen of the bowel. This ulceration may occur either before operation and drainage or afterward. What happens to the abscess if it is not opened and drained? What became of the appendiceal abscesses before we operated for appendicitis?

INTERN: They were fatal to the patient.

DR. MURPHY: Often, but not always. If they were always fatal, none of you would be attending my clinic today, for I had

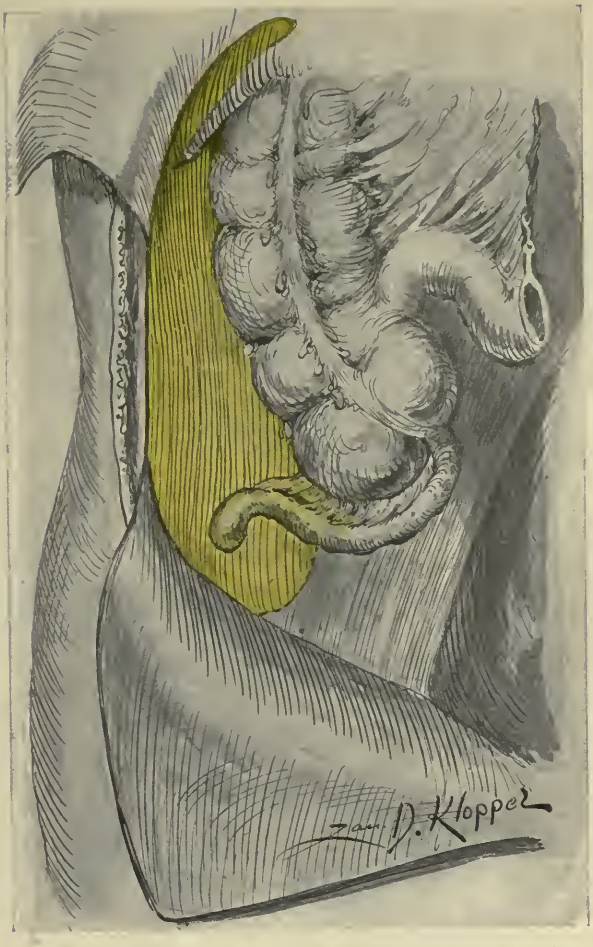


Fig. 1.—Perforation of the appendix and secondary abscess formation to the right of the cecum and ascending colon. This diagram shows the route which such an abscess pursues in its upward spread and how easily it may form a perirenal abscess or a subphrenic abscess. The pus is represented here as having spread beneath the diaphragm (from Dr. Murphy's article in Keen's "Surgery").

three attacks of appendicitis myself in the preoperative era, one, at least, with pus formation; but I am here to-day, and why? Because the abscess ruptured into the bowel after ulcerating through its wall, and thus underwent spontaneous evacuation and resulted in recovery. Nine, ten, or eleven days after the formation of the abscess one looks for what? For the appearance of offensive-smelling pus in the stools, the peculiar odor for which the nurses and even the housewives and the mothers learned to look in that era. Exactly the same pathologic process still goes on in the cases of appendiceal abscess that are allowed to go unoperated for a number of days, which we see so often following the so-called "expectant" or "dry" treatment of appendicitis.

The mention of "expectant treatment" for appendicitis to me is like the waving of the *banderillero's* red scarf at *el toro*. I can't keep quiet. I am still hunting for a term which expresses my opinion of the method. I find the English vocabulary too limited, however, to suit my desires. A Latin medicus might have called it the *expectans mortem* treatment. To name it the *mañana* method is not malapropos, but it is too mild. To describe it as *dolce far niente* is expressive, but this soft Italian phrase is impossible when I am showing all my teeth in a Rooseveltian glare. If I had Roosevelt's genius for phraseology I might find the needed term, but as yet it eludes me.

When we operate these late abscesses they are often just on the point of rupturing into the bowel, and the operation fails to prevent the perforation. The abscess ruptures into the bowel and some of the bowel contents escape into the abscess cavity and then out through the drain opening, making a fecal fistula. Putting large gauze packs into the abdomen in the presence of a virulent infection may lead to a fecal fistula, but we do not use these large packs or Mikulicz tampons now; that procedure is as ancient history as is the plan of waiting until the fifth or sixth day before operating. We surgeons do not operate patients with pus outside of the appendix when we can avoid it. But when we *do* have to operate under such circumstances, that fact alone is incontrovertible proof that the case has been badly managed up to that time. No case should be allowed to go until an abscess has formed outside of the appendix. The abscess may burrow where? What are the directions in which an abscess may burrow when starting from the appendix?

Intern: It may burrow upward or downward.

Dr. Murphy: Tell me exactly where and why? Why do these abscesses burrow? Let us see how much you know about them. We see them in this hospital almost every day in some one or other of the clinics.

Intern: The abscess may spread upward and involve the gall-bladder, or it may descend into the pelvis and produce a pelvic cellulitis.

Dr. Murphy: No! No! Not at all. You are shooting bravely, but your gun is loaded with junk. A burrowing appendiceal abscess does not indiscriminately scatter about like that. Concentrate your fire more. Tell me the directions and ranges, and why? Remember that the normal position for the appendix is a little lower than McBurney's point. The appendix may lie where else? It may be turned backward and upward on the posterior surface of the colon or on the external surface of the caput coli. If an abscess forms around an appendix lying in this location, where does it burrow? Where can it burrow? Which is the direction of least resistance? It burrows upward along the ascending colon, and becomes a perirenal abscess or a subphrenic abscess. Finally, it may even perforate the diaphragm into the pleura, then into the lung, and be expectorated by the patient. The offensive expectoration may make the diagnosis of subphrenic abscess in a doubtful case when a lung abscess and an interlobar empyema can be ruled out.

The appendix which hangs over the brim of the pelvis often makes annoying adhesions and dangerous complications when it becomes inflamed. We operated a case last summer in which the appendix hung over the brim of the pelvis and was adherent to the iliac vein and artery. These vessels were pulsating in the field of operation. I was very careful in carrying out the dissection, because of the ever-present danger of tearing a hole in the artery or vein while dislodging the tip of the appendix, which I finally succeeded in doing without accident. If one is not mind-

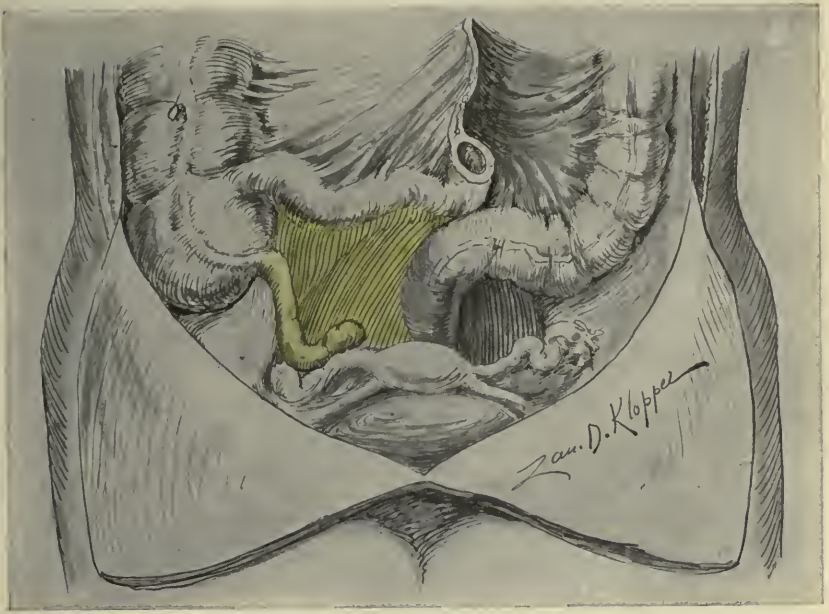


Fig. 2.—Perforation of an intrapelvic appendix adherent to the right ovary and tube, and secondary intrapelvic abscess formation on the right side. Note that the abscess is bounded above by the ileum and its mesentery, on the left by the rectum and sigmoid, below by the uterus and adnexas, and to the right by the cecum (from Dr. Murphy's article in Keen's "Surgery").

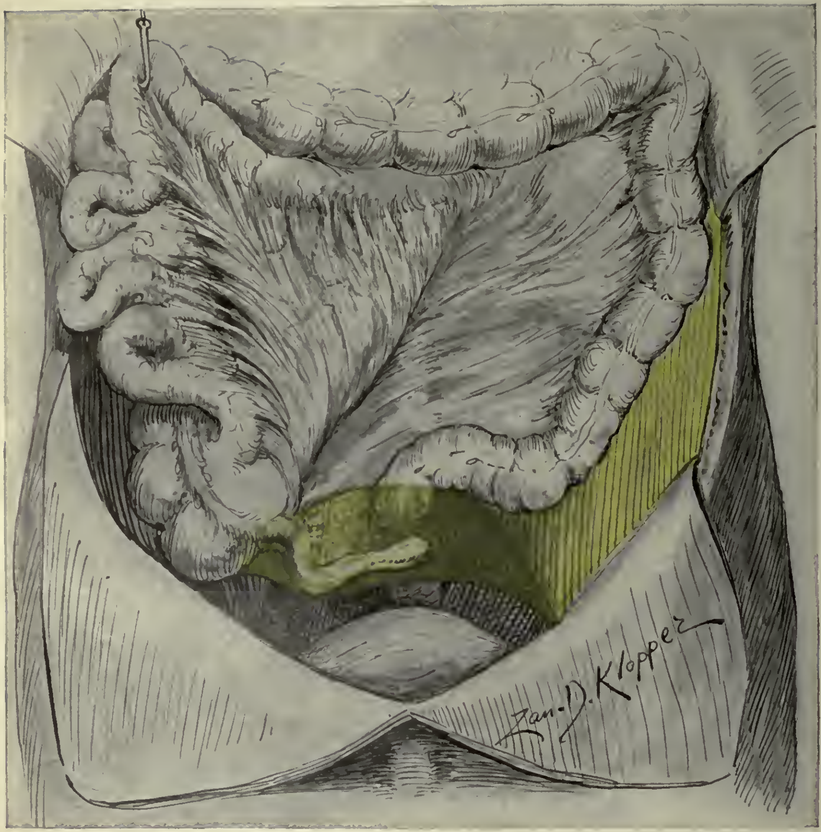


Fig. 3.—Showing how an intrapelvic appendix which lies across the rectum may produce by its perforation a left-sided pelvic abscess which tends to rise out of the pelvis and point near the left anterior superior spine. Such an abscess is bounded on the right by the rectum, sigmoid, mesosigmoid, and descending colon (from Dr. Murphy's article in Keen's "Surgery"). This illustration and the following one illustrate the necessity of opening the free peritoneal cavity to determine the relations of the abscess to the large bowel before draining the abscess.

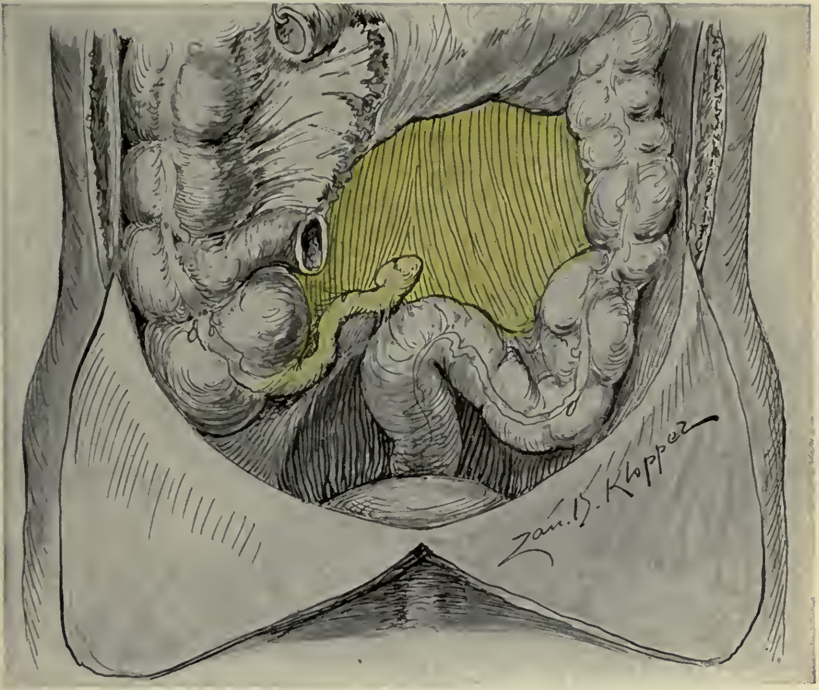


Fig. 4.—Perforation of the appendix and secondary abscess formation in the space bounded on the right and above by the cecum and the root of the great mesentery, and on the left and below by the sigmoid and the descending colon (from Dr. Murphy's article in Keen's "Surgery").

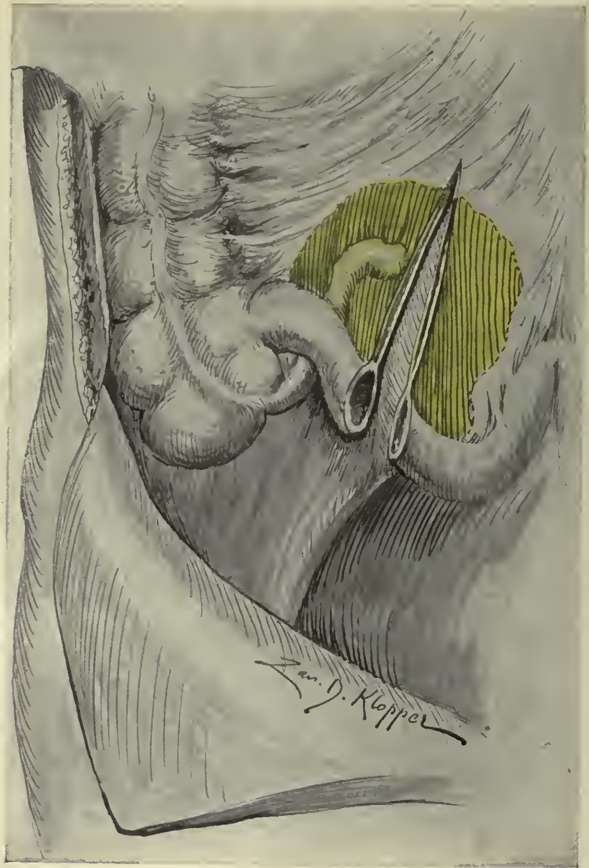


Fig. 5.—Perforation of an appendix lying between the two layers of the great mesentery, with secondary abscess formation inside the mesentery. To drain such an abscess the mesentery must be split open. The danger of vascular complications in such cases is naturally great (from Dr. Murphy's article in Keen's "Surgery").

ful of the fact that not only the vein, but also the artery, lies in the field of danger, one may tear a gaping hole in the artery, as Israel had the misfortune to do in the early days of the appendicitis era. He was a master surgeon. Undismayed by the accident, he compressed the vessel digitally and *kept* his finger on it while he cleaned out the field of operation and enlarged the abdominal opening with his other hand, and packed off the involved area from the rest of the peritoneal cavity; then he sutured the artery successfully. Most men in those days would have performed a ligation and would have run the risk of causing a gangrene of the leg. That was the first successful suture of the internal iliac artery ever performed.

If an abscess forms from an appendix which hangs over the pelvic brim, the pus collects in the pelvis. The abscess may remain circumscribed in the pelvis, but it can and not infrequently spreads upward out of the pelvis. When the sigmoid and rectum limit it on the left, the abscess passes upward over the right iliac vessels and comes to lie in the right side of the lower abdomen. The sigmoid often lies so far to the right side of the median line that the appendices epiploicæ have been excised innumerable times when it was intended to remove the appendix vermiformis. Infiltrated and covered with pus, these structures look very much like an appendix vermiformis. Such patients often have a subsequent recurrent attack of their appendicitis, and the doctor, if he is wise, then begins to wonder if it was really the appendix which he cut off. Such an error can be easily avoided by having an assistant examine the supposed appendix as soon as it is removed. I well remember a man whom I operated for a second attack of appendicitis "after the appendix had been removed" by another surgeon. We had much difficulty in convincing the patient that a deliberate fraud had not been practised on him by the first operator.

Sometimes the pelvic abscess passes across the rectum and forms a second abscess to the left of the rectum; or when an exceptionally long appendix lies far down in the pelvis, its tip may lie across the rectum, and if perforation occurs in the tip, the first abscess may form on the left side of the pelvis. Such left-sided pelvic abscesses, if not drained promptly, may spread upward, being bounded on the right by the rectum and sigmoid. They tend to point just to the inner side of the left anterior superior iliac spine, bearing exactly the same relation to the left anterosuperior iliac spine that an ordinary appendiceal abscess usually does to the right spine.

In opening these secondary abscesses on the left side of the abdomen the location of one's incision is a matter of great importance. It is essential to determine where the descending colon and sigmoid lie—that is, whether the abscess is on the lateral side or the medial side of the large bowel. If one cuts down directly on such an indurated mass, which is adherent to the abdominal wall, there is serious danger of actually cutting through the bowel in entering the abscess, a *contretemps* which is very likely to be fatal.

The proper and only safe procedure is to enter the free abdominal cavity on the medial side of the abscess, inspect the field, and determine the position of the colon and sigmoid, and then open the abscess. The same opening of the free peritoneal cavity is necessary in operating on an intestinal fistula having such an abscess for its cause, and the reason for so doing is the same.

These left-sided appendiceal abscesses may spread upward along the outer and posterior surfaces of the descending colon, just as the ordinary retrocecal abscess spreads upward on the right side; and one sees occasionally a left-sided perirenal abscess of appendiceal origin which has followed this route.

INTESTINAL FISTULAS

I. Congenital fistulas:
  1. Umbilical
    a. Strangulation of the intestine during ligation of the cord.
    b. Persistence of the vitelline duct.
  2. Ventral—median—congenital occlusion with perforation.
  3. Vesico-intestinal
  4. Vagino-intestinal
  Urethro-intestinal
    a. Defect of development.
    b. Imperforate anus with ulceration and perforation.
  5. Post-rectal:
    a. Congenital sinus.
    b. Infection of postrectal dermoids.

- II. Operative fistulas:
  - A. Intentional:
    - 1. Artificial anus (permanent)
      - a. For innocent obstructions.
      - b. For malignant obstructions.
    - 2. Artificial anus (temporary).
      - a. For subsequent resection.
      - b. For temporary obstruction.
      - c. Puncture for the relief of distention.
      - d. To give the bowel physiologic rest.
    - 3. Artificial anastomoses
      - a. Gastro-enterostomy.
      - b. Entero-enterostomy.
      - c. Cholecystenterostomy.
  - B. Accidental:
    - 1. Laceration of the intestine—separating adhesions.
      - Recognized or unrecognized
        - a. Inflammatory.
        - b. Neoplasms.
    - 2. Impairment of the wall—subsequent perforation.
    - 3. Division with the scalpel or scissors.
    - 4. Compression with:
      - a. Forceps.
      - b. Ligature.
      - c. Suture.
      - d. Drainage.
      - e. Packing.
    - 5. Curetment, by perforation of the uterus and wounding of a loop of bowel with the curet.
    - 6. Criminal abortion:
      - a. Direct by penetrating uterine wall and intestine.
      - b. Indirect—peri-uterine perforation.
    - 7. Vagino-intestinal perforation:
      - a. Caustics and pastes.
      - b. Pessaries.
      - c. Vaginal hysterectomies.
      - d. Puncture of a vaginal hernia.
    - 8. Failure of union after enterectomy and enterorrhaphy due to—
      - a. Infection.
      - b. Defective coaptation.
      - c. Intestinal ischemia.
      - d. Operations on a diseased intestinal wall:
        - a. Malignant neoplasms.
        - b. Mural
          - Gangrene.
          - Infection.

- III. Pathologic fistulas:
  - 1. Mural ulceration and perforation due to:
    - a. Malignant neoplasms.
    - b. Tuberculosis.
    - c. Actinomycosis.
    - d. Typhoid.
    - e. Pressure from foreign bodies:
      - a. Bodies introduced from without.
      - b. Gall-stones.
      - c. Enteroliths.

        - f. Ulceration of the intestine above a stricture—the latter due to:
            - a. Syphilis.
            - b. Tuberculosis.
            - c. Dysentery.
            - d. Congenital stricture.
        - g. Duodenal ulceration.
        - h. Syphilis.
    - A. Opening externally.
    - B. Opening internally:
        - a. Stomach.
        - b. Kidney and ureter.
        - c. Entero-intestinal.
        - d. Bladder.
        - e. Female genital tract.
        - f. Diaphragm, pleura, or lung.
- 2. Fistulas the result of intestinal strangulation:
    - a. Intra-abdominal
        - 1. Hernia.
        - 2. Meckel's diverticulum.
    - b. Extra-abdominal.
- 3. Perforation from diseases of neighboring structures:
    - A. Malignant: carcinoma of neighboring viscera.
    - B. Non-malignant:
        - a. Gastric ulcer.
        - b. Pressure necrosis:
            - a. Gall-stones
                - Gall-bladder.
                - Cystic duct.
                - Common duct.
            - b. Nephritic calculi.
            - c. Ureteral calculi.
            - d. Vesical calculi.
        - c. Infectious cholecystitis:
            - a. With calculi.
            - b. Without calculi.
        - d. Nephritic abscess:
            - a. Pyogenic.
            - b. Tuberculous.
            - c. Nephritic calculi.
        - e. Perinephritic abscess:
            - a. Pyogenic.
            - b. Tuberculous.
        - f. Diseases of the ureter:
            - a. Tuberculosis.
            - b. Calculus with infection.
        - g. Hepatic abscess.
        - h. Osteitis:
            - a. Tuberculous.
            - b. Osteomyelitis of spine and ilium.
        - i. Infected ectopic gestation.
        - j. Dermoids—infected.
        - k. Salpingitis:
            - a. Purulent.
            - b. Tuberculous.

l. Ovarian cysts—torsion of the pedicle with auto-infection.
m. Vesical tuberculosis.
n. Peri-urethral (infections) vesicorectal } fistulas.
Prostatic (infections) urethrorectal } fistulas.
o. Rectal (infections).
Perirectal (infections).
p. Phlegmon of the abdominal wall.
q. Ulceration of the skin over a hernia, with perforation of the contained bowel.
r. Pelvic abscess:
  a. Tuberculous.
  b. Pyogenic.
  c. Urinary infections.
s. Echinococcus cysts of various neighboring structures.
4. Peritonitis:
  A. Local.
  B. General.
IV. Traumatic fistulas:
1. Gunshot wounds.
2. Stab and perforating wounds.
3. Contusions:
  a. Kicks and blows.
  b. By foreign bodies.
  c. By being driven over.
  d. Falls.
  e. Fractures of bones.
V. Postparturient:
1. Pressure necrosis from long retention of the infant's head in one spot.
2. Laceration.
  a. Entero-vaginal.
  b. Entero-uterine.
3. Through rupture of the genital tract in operative deliveries.
4. Amputations of a knuckle of bowel for a supposed funiculus (cord).

[This table is a copy of the chart used in Dr. Murphy's clinic to teach the undergraduates the possible sources of intestinal fistulas. To the practitioner, when a complicated fistula comes to him, a few minutes' perusal of this chart may be of decided service.—Ed.]

In the classification of fistulas which we have had placed on the blackboard you will note that the right iliac fossa is the most common location. A not uncommon type of fistula that occurs in the left iliac fossa is that which follows a diverticulum of the sigmoid. An abscess sometimes forms here after recurrent attacks of peridiverticulitis, and a mass of induration develops, usually, around the sigmoid. When one opens such an abscess, which always has a direct communication with the lumen of the bowel, a fecal fistula is particularly prone to follow. Not only at operation, but even at autopsy, it may be difficult to convince oneself that these are not malignant lesions on account of their great induration and hardness; for the amount of connective-tissue formation is often out of all proportion to the small amount of pus present. When one resects the diverticulum and the bowel and reaches the mesentery of the sigmoid, one may find the diverticulum taking its origin from a small perforation of the wall of the sigmoid where some of the feces were in a fold of the wall and burrowed their way along the wall, much as a *fistula in ano* starts in one of the rectal pockets and burrows along and outside the wall until finally it develops into a full-fledged fistula, with a cutaneous as well as a rectal opening.

FECAL FISTULA FOLLOWING A LATE OPERATION FOR PERIAPPENDICEAL ABSCESS BURROWING UPWARD BEHIND THE CECUM.—EXCISION AND CURE

HISTORY

The patient, a married woman twenty-nine years of age, entered the hospital in June, 1914. Her family and past history is negative. Her present trouble began in October, 1912, when she was suddenly seized with severe, cramp-like pains in the lower right quadrant of the abdomen. The patient kept the right leg flexed as she lay in bed, because so doing somewhat allayed the pain. Immediately she had high fever, nausea, vomiting, and much tenderness over the right lower quadrant. In about five days this tenderness shifted to the back. There was so much pain and local tenderness that she would not allow any one to touch the part, even gently. She was brought to the hospital October 29, 1912, ten days after the onset of the pain, and operated immediately. Pus was found in the free peritoneal cavity, and a pocket also extended upward behind the cecum.

Since the operation the wound has never closed, but has continued draining constantly—so copiously, in fact, that it has to be dressed three times a day. She complains of a stinging pain over the area, especially marked at her menstrual periods.

COMMENTS AND OPERATION

DR. MURPHY: What was done at the operation?

INTERN [reading report]: An incision was made over the right lumbar region, where there was an area of infiltration and edema. This proved to be due to an abscess located further in front. The abscess extended down only to the subperitoneal cellular tissue, and was packed with a single strand of nosophen gauze. Then the patient was turned over on her back and an anterior rectus incision was made somewhat higher up than the usual appendix incision. There was some edema found in the abdominal wall also. The peritoneum was white and thick. The peritoneal cavity contained a considerable quantity of foul-smelling pus, apparently originating at the appendix. Two rubber tubes and a strand of nosophen gauze were inserted for drainage. The abscess had also burrowed along the colon to beneath the diaphragm. The sponges were counted and reported to be correct.

DR. MURPHY: This was an appendicitis case that had been allowed to go to the tenth day without operation; and the abscess had burrowed upward. It was of the type which I described in my article in Keen's "Surgery" (Vol. IV, p. 745, Fig. 417) as burrowing on the outer side of the colon up into the subphrenic space, as well as involving the pericecal neighborhood. She had an area of inflammatory edema in her loin so marked that we opened the abscess first from behind, and then, when we discovered the origin of the abscess, turned her around and made the usual drainage opening in front. It was a typical appendiceal abscess with areas of gangrene of the wall of the intestine. Ever since the operation she has had a fetid and purulent, but not a distinctly fecal, discharge.

This case was treated at the onset on the "expectant" plan, and she went from bad to worse. Dr. Golden was induced to go out and see the patient after I had declined to take the case, saying, "Let her doctor carry the case to a finish on the 'expectant' plan, since he started it." Dr. Golden, after using some persuasion, induced the patient to come to the hospital, and then threw the responsibility of the case on me when he found in what desperate straits she actually was. She seemed practically moribund at the time when we operated. Her own doctor would have kept right on treating her on the expectant plan when he had nothing more to expect except an autopsy if her people would have let him. He could no longer hold the case, so he sent for us to write the death certificate. Dr. Golden, in his usual genial spirit of helpfulness, answered the S. O. S., but when he saw the reefs, he sent out a call on his own account. We operated and were rewarded with an unexpectedly favorable outcome; but this little fistula, the result of the intestinal necrosis present, has failed to heal up to the present time. We did not remove the appendix at the operation, but were content simply to open the abscess cavity in front and behind and drain it thoroughly.

A question which has rather concerned us is whether we have to deal here with a simple sinus containing a foreign body somewhere in it, keeping up the suppuration, or whether this is a fecal fistula connecting with the bowel. The discharge is purulent, rather than fecal; but Dr. Hochrein, who has dressed the patient the greater part of the time, states that after meals containing berries he has seen berry seeds appear in the discharge from the fistula on several occasions. Dr. Golden, who has done fewer of the dressings, has not seen the seeds; but in such a case we must draw our conclusions from the man who has seen the seeds, rather than from him who has not seen them. The berry-seed test, in my hands, has been the most reliable of all tests as a means of determining whether we are dealing in such a case with a fecal fistula or not.

[In attacking this fistula Dr. Murphy followed his usual technic, which has been previously described in the CLINICS, first opening into the free peritoneal cavity, exploring it, isolating the fistula and the loops of bowel adherent to it, separating them from their attachment to the anterior abdominal wall, and from their attachments to one another, closing the intestinal perfora-

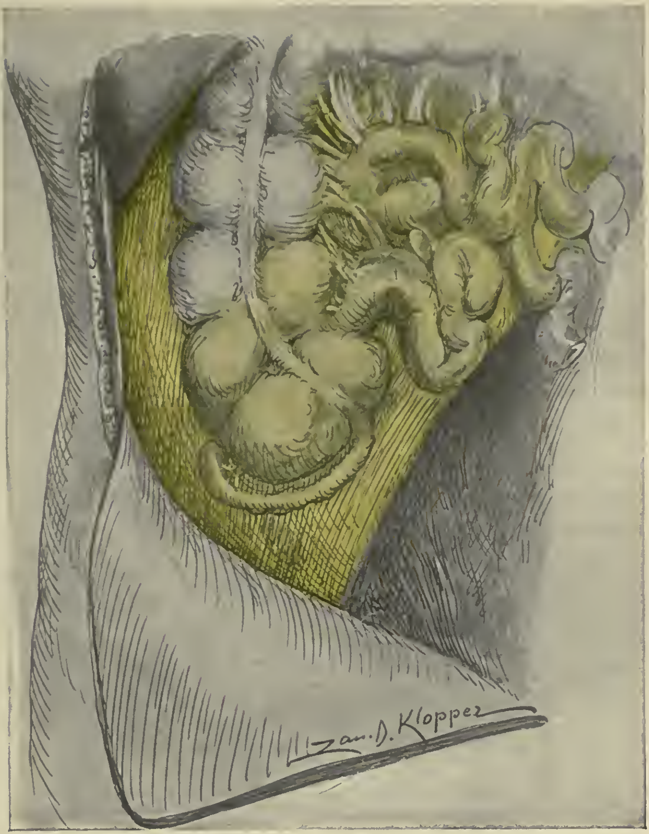


Fig. 6.—Localized fibrinopurulent peritonitis from a ruptured appendix lying to the outer side of the cecum. Note the abscess spreading upward along the outer side of the cecum and ascending colon to the under surface of the liver. Note the involvement of the head of the cecum and the terminal coils of the ileum in the exudate (from Dr. Murphy's article in Keen's "Surgery").

tion present, and covering over all raw surfaces before closing the wound down to the drainage opening.—Ed.]

Let the record show that the fistula led upward along the outer side of the colon toward the right subphrenic space; that there were several coils of intestine adherent to it, or adherent to one another around it, although only two coils were really closely united with it; that the gall-bladder was exposed early in the operation, and freed from adhesions to the wall of the sinus, so high up did the latter run; that the transverse colon with the omentum was freed from its fixation to the fistulous zone; that the ascending colon was freed from the fistula which lay between the colon and the anterior abdominal wall, a difficult procedure technically, although finally carried through successfully without opening the intestine; that the colon after being freed from the abdominal wall was swung forward and upward and carefully inspected, and that beneath it, at the end of the sinus, was found a hard concretion resembling a gall-stone; that the cecum was then turned up and the appendix found lying behind it near the end of the fistula; that the appendix had nearly undergone spontaneous amputation close to its base at the apparent site of its rupture; that just distal to the old perforation the appendix contained a firm, hard calculus, almost identical in appearance with the one previously found lying free in the fistula; that we amputated the appendix, using catgut for the ligature of the appendix, as well as for the purse-string and Lembert stitches, because this is an infected field and very likely to suppurate, and we never place unabsorbable sutures, such as Pagenstecher's linen, in a field of suppuration; that we also found a perforation from the ascending colon into the fistula just below the gall-bladder, and that we closed this opening with mattress Czerny stitches of Pagenstecher linen and Lembert and mattress stitches of iodized catgut; that the first concretion (gall-stone?) seen lay rather close to this opening; that we excised the fistula and covered all the raw surfaces exposed before closing the wound down to the small drain, which ran to the stump of the sinus, at the bottom of the field of operation; that the superficial wound and subcutaneous fat were sponged off with a 0.5 per cent.

lysol solution, in order to destroy any colon bacilli which may have escaped into the wound, and which are very liable to induce a suppuration in the subcutaneous fat, if allowed to remain there.

The lysol seems to have a stimulating action on tissue repair as well as an antiseptic action. We exposed the right kidney during our final inspection of the abdomen, and found it entirely uninvolved in the old inflammatory process, although lying very close to the latter. It evidently played no rôle in the causation of the suppuration, nor has it been affected by the latter.

Let the record further show that I completed the closure of the abdominal wall personally; that there is likely to be a hernia here on account of the extensive dissection of the abdominal wall, which was found necessary, and on account of the extensive area of scar tissue excised from the abdominal wall, before we were able to secure normal tissues to approximate to one another; that we sutured the rectus aponeurosis accurately for the entire length of the incision, except for the small opening where the drainage-tube was inserted.

When the patient returns to her room we shall let her lie flat on her back, elevating neither the head nor the foot of the bed.

What is the prognosis here? It is good, for this reason—and the Germans express it very aptly—"*Unkraut vergeht nicht!*" A Hibernian translation—I suppose it must be—runs: "Stuff which is not cabbage won't quit"—either Eden or the Auld Sod. "Weeds never die!" These fecal fistula cases, especially the tuberculous ones, pus and pus and are operated and reoperated. One would think they would die, but the greater their misery, the tighter they seem to hang on to life—a burden to themselves and their friends, a source of despair to their doctors, and a reproach to our vaunted medical science.

The greatest danger in the present operation is not from peritonitis, but from pneumonia. In much of the work in the abdomen, particularly that in the upper abdomen, pneumonia is our greatest bugbear and the principal source of our mortality.

The difficulty and danger of these operations depend largely on the number of the perforations of the intestine into the fistula. If there is no opening at all into the bowel, one cannot speak of the case as one of fistula, but as a sinus. A sinus is usually kept open by some foreign body—an unabsorbable ligature, a concretion, or a calculus—such as we found in this case—a forgotten sponge or instrument, or even exuberant granulations. The simple removal of the foreign body allows the sinus to close. In other cases the openings from the fistula into the bowel may be extremely numerous, and I well remember one of my earliest cases of this kind, and also one of the worst that I have ever seen, a woman who had 25 openings from the small bowel into the common cloaca of the fistula. Dr. Franklin Martin referred me that case. I cannot recollect ever thanking him for it. I don't know that any of you would have thanked him had you been in my place. I have heard it said that "Any fool can hang on, but it takes a canny man to know when to let go." When Dr. Martin let go of that patient he established a reputation with me of being one of the canniest of the profession; and I have never had any reason since that time to lower my estimate of his sagacity one iota.

It would not take many cases of appendicitis like the present one, I think, to make a convert to the early operation idea of even the most hardened advocate of the expectant treatment. The surgeon who always operates early never has any human wrecks like this woman hanging like a millstone on his conscience and his reputation.

June 30, 1914: There is no pus at all coming out of the drainage opening. The silkworm-gut stitches have been removed. There is a perfect primary union of the skin. The drainage-tube has been removed, but the horsehair stitches still remain.

July 7, 1914: There is a complete primary union of the entire wound—a perfect result. The horsehair stitches have been removed. The patient will sit up every day from now on.

[Note.—The patient made a complete and perfect recovery. —Ed.]

FECAL FISTULA FOLLOWING PERITONITIS, PROBABLY OF APPENDICEAL ORIGIN.—EXCISION OF FISTULA. —SEPARATION OF ADHESIONS.—RESECTION OF TWELVE INCHES OF SMALL BOWEL.—UNEVENTFUL RECOVERY

HISTORY

The patient, a married man forty-six years of age, entered the hospital March 22, 1914. His family history, past history, and venereal history are all negative. He has been constipated for twenty years.

His present trouble, as near as he knows, began about April or May, 1908. At that time he noticed a feeling of distress about one hour after heavy meals, and sometimes a sensation of heaviness as if there were a large mass present in the stomach. He belched much gas from this time on, and had rather frequent eructations of acid fluid. This condition was usually most marked at the times when he was working the hardest (no reference to season). He also found that heavy foods, meats, and cold salads distressed him much more than warm and light foods. He was not nauseated, nor did he vomit at all in 1908.

This "dyspepsia," with exacerbations, continued more or less constant, gradually becoming more severe, until one day in March, 1912, he had a very severe attack. The epigastric pain at this time came on about one hour after breakfast and continued to increase in severity until by noon it was sharp and cutting in character. He ate no luncheon. At 2 P. M. he passed a stomach-tube on himself and washed out his stomach. He immediately felt better; but one hour later a chill came on which lasted ten minutes. He went to bed and perspired freely. On the next day he was up and about the house, but ate very little. He was very tender over the epigastric region, and he could locate a definite tender area there about the size of the palm of the hand. In this attack he did not vomit, but he felt nauseated.

From this time on he had more frequent and more severe attacks, which he found could frequently be aborted by eating something. When they were too severe for eating to ameliorate the distress, he could always get relief by pumping out his stomach with a stomach-tube, a procedure which he preferred to the induction of emesis.

About January 23, 1914, at midnight, he was awakened with sharp, cramp-like pains occurring at about the level of the umbilicus, and radiating downward toward the bladder and into both iliac fossæ, being most marked in the right side. He felt nauseated immediately, and vomited a greenish material one hour after the onset of the pain. Half an hour later he had a chill which lasted ten minutes, and was followed by profuse perspiration. He had had no pain at all that night when he retired. Two hours after the onset of the attack he took an enema and felt much better after evacuating it, although the pain continued severe until the following evening. He knows nothing regarding his temperature at this time. The next evening, because of persistence of the pain, he called in a doctor, who gave him a hypodermic injection of morphin, but made no diagnosis. He advised the patient to come to his office for further examination as soon as he felt able. During this attack the patient was tender all over the lower half of his abdomen, and especially about the bladder region. His abdomen was somewhat distended. He remained in bed two days, and after getting up did no active work for six days, but remained in the house. Then he went to consult the doctor. All this time the dull pain and also the tenderness had continued present in the lower abdomen. His appetite was very poor, and his bowels were constipated.

On January 30, 1914, the doctor examined him, diagnosed appendicitis, and advised an immediate operation. The patient went to the hospital the next day, and was operated on the day following. On opening the abdomen the doctor found the whole lower abdomen filled with pus. On further examination he discovered a perirectal abscess, he stated, which seemed to be independent of the other condition and was walled off by adhesions between the rectum and bladder. The doctor thought that this perirectal abscess was the result of inflamed hemorrhoids.

The hemorrhoids have been noticed by the patient since he was eighteen years of age, but they rarely bothered him until during the past four years. Since then they have pained him considerably while he is working and when his bowels move. For the past six months they have bled quite frequently.

Two large rubber tubes were inserted by the surgeon for drainage at the lower angle of the abdominal wound. The appendix was found bathed in pus and "looked very bad," but was not ruptured and was not removed.

Five days following this first operation the patient's abdomen was opened again because of an obstruction in the bowel involving the ileum for about nine inches from the cecum. This portion of the bowel was resected and a loop of ileum further back was brought over to the cecum and an anastomosis with the Murphy button was made. There was no fecal discharge at this time, but the abdomen was filled with pus. The patient did not seem to improve much after this procedure, but his bowels moved a few times. About March 8 feces began to come through the skin incision. His abdomen was then opened and another attempt was made to repair the intestines. Three holes in the newly anastomosed ileum were closed. Around the button in the cecum there was a leak. His condition gradually became worse, and so he was brought here to Mercy Hospital.

COMMENTS AND OPERATION

Dr. Murphy (June 29, 1914): What about his temperature?

Intern: The patient does not know anything about his temperature.

Dr. Murphy: That fact should be recorded in the history. It is not in there.

Dr. Murphy: How many days after the primary operation was he operated the second time?

Intern: Five days.

Dr. Murphy: From the history, the primary lesion in this case appears probably to have been an appendicitis with a pericecal abscess.

[This operation was a severe and extensive one, and it took the unusually long time of nearly two hours to complete. Dr.

Murphy first blocked the fistula by filling it tightly with narrow strips of iodoform gauze and, in addition, clamping it with a couple of volsellum forceps. The peritoneum was opened outside the line of intestinal and omental adhesions to the anterior parietes, the free peritoneal cavity being found at the first attempt. Before attacking the line of adhesions the peritoneal cavity was explored in order to locate the seat of the trouble more accurately. The gall-bladder was found free from stones and other pathologic changes, and the pylorus was without induration and freely patent. The pylorus and duodenum were examined with special care, because the early portion of the patient's history suggested the possible existence of a duodenal ulcer. The mass of adhesions surrounding the fistula appeared to consist of only cecum, ileum, and omentum, thus pointing distinctly to the appendix as the source of the trouble.

The next step was to free the attachments to the anterior parietes of all the intestinal coils involved in the fistula, so that the latter, together with the surrounding coils, could be brought outside the abdomen, and made more accessible for the carrying-out of the subsequent steps of the operation. The loops of bowel were then picked up one by one and separated from the general mass. All raw surfaces on the detached coils were covered over either by invagination or by approximation to a neighboring loop. The coils with an opening into the fistula were separated last, and each opening closed with a purse-string suture of Pagenstecher linen, followed by one or two catgut Lembert stitches.

One loop of bowel contained within a short area three openings into the common cloaca of the fistula. On account of these triple perforations in close proximity it was deemed advisable to resect the loop for a distance of 12 or 14 inches, rather than run the risk of leakage occurring or a stricture developing in this weakened area. An end-to-end anastomosis by the suture method was performed, the Czerny stitch being of Pagenstecher linen and the Lembert of catgut. The parietal wound was closed layer by layer, as usual, leaving a small rubber-tube drain in the lower wound angle. After the suture of the peritoneum and fascia the subcutaneous cellular and fatty tissue was swabbed off with a sponge wet with 5 per cent. carbolic solution, in order to destroy any colon bacilli present.—Ed.]

These fistula cases are always severe and disagreeable. We have spent nearly two hours on this work, and a very tedious two hours it has been, as I think my audience will agree. It sometimes seems to me as if I do not have much to show after these operations, in proportion to the amount of time and energy expended. But if the fistula stays closed and no peritonitis develops, the patients are always greatly pleased, and that is our reward. However, taking them all in all, they are not pleasant cases to handle. They are the penalties one pays for being notorious. We had two more of them last week, both of whom, I am happy to say, are making an afebrile and uneventful recovery.

This patient immediately on his return to bed will be placed in a high Fowler position. He will have nothing by mouth for five days. We shall give him a pint of water and a little sodium bicarbonate per rectum every three hours. These enemata will furnish him fluid enough. We shall not administer salt solution. At his age—forty-six years—sodium bicarbonate solution is better borne than saline.

[Note.—Dressing, July 10, 1914—Dr. Murphy. The tube was shortened two inches. There is not the slightest odor or discharge from the wound; therefore we are certain that we have secured an intact intestinal union. On July 14 we shall begin to take out the silkworm-gut stitches, beginning from above.]

[The patient made a complete and uneventful recovery.—Ed.]

APPENDICEAL CONCRETION PRODUCING ULCERATION, PERFORATION, AND ACUTE PERITONITIS.—GANGRENE OF CECUM.—POSTOPERATIVE FECAL FISTULA.—OPERATIVE RELIEF

HISTORY

The patient, a male, aged thirty-seven years, entered the hospital July 9, 1914. His family history is negative as to tuberculosis and cancer. He had typhoid fever without complications twelve years ago. He had diphtheria thirty years ago. He denies venereal infection.

The present trouble dates back to June 10, 1913, when the patient was suddenly taken ill about 3 P. M., with severe cramping pains in the right lower quadrant of the abdomen. He did not vomit until the next morning. He then had a temperature of 103° F., with tenderness and rigidity over the right lower quadrant of the abdomen. Eighteen hours after the onset of the pain he was taken to a hospital and operated for appendicitis. He does not know whether or not his appendix was removed, but a report from his physician states that the appendix was found ruptured, that it contained a fecal concretion, and that the head of the cecum was found gangrenous. The abscess was drained with a rubber tube for five days. Eight days after the operation the patient first noticed fecal material draining from the wound. Two months later an indurated area appeared on the right side, in the neighborhood of the wound, causing him pain. This proved to be an abscess. It was opened and a rubber tube drain inserted. Two days later this opening also began to drain fecal material.

On December 13, 1913, the patient was operated again, the incision being made through the drainage opening on the right side. The doctor told him immediately after the operation that one leak was closed; but two days later fecal material again drained from the previous site. On March 28, 1914, an incision was made at the site of the primary incision, but a little higher up, and eight inches of the small intestine were resected and a lateral intestinal anastomosis made. A leak in the large intestine opposite the ileocecal valve was also closed. In six or seven days, however, fecal material again drained through the wound.

The patient states that feces are passed per rectum about once a week. He has to use enemas to secure a movement, and if he is lying down when the enema is taken, the latter runs out of both wounds in the abdomen.

He has lost about 65 pounds in weight since the first operation. He has no cough, no afternoon or evening rise of temperature, or night-sweats. The Koch tuberculin test was made and proved negative.

COMMENTS AND OPERATION

DR. MURPHY (July 11, 1914): This is one of four cases of fecal fistula in the hospital on our service at the present time, three of them the sequences of the "expectant" treatment of appendicitis. This story is somewhat different from the average in such cases, and we have tried to figure it out.

It is extremely unusual to have a fecal fistula develop following an appendectomy performed within eighteen hours after the onset of the appendicitis. It is the cases which are operated late, the victims of the "expectant" or "dry" treatment of appendicitis, who so frequently develop this very unpleasant complication. In an early case there must be a specific and probably elicitable cause of an unusual nature to produce an escape of fecal contents. Either that is the case or the history is wrong. The latter is not infrequently the explanation. The patients have a mild onset of their appendicitis, the appendix becomes gangrenous and therefore painless, and when the pain of the beginning peritonitis sets in, the patients may insist that the latter was the beginning of the trouble, whereas they should have dated the onset two, three, or four days earlier, when they first felt a little diffuse abdominal pain and were nauseated for a short time.

Just before the American Medical Association meeting in 1912 a man came over to my office from his work complaining of sudden abdominal pain which had compelled him to stop work. He had the nausea, fever, tenderness, and leukocytosis, so we took him over to the hospital immediately and operated, as we thought, within the first two or three hours of the attack. We were greatly surprised at operation to find a completely gangrenous appendix. But when we came to question him closely after the operation he admitted that he had had some abdominal discomfort on the preceding day, but that it was so slight in comparison with the pain of his beginning peritonitis that it hadn't occurred to him to mention it.

About all we know about to-day's patient is that his appendix had already ruptured at the time of this relatively early operation, and that he had free pus in his peritoneal cavity.

INTERN: The patient's physician is here. He knows all about the case.

DR. MURPHY: Did the patient have any symptoms like a beginning appendicitis three or four days preceding the severe attack of pain?

PATIENT'S PHYSICIAN: No, he did not. He worked until 3 P. M. of the day before he was operated.

DR. MURPHY: The next question to ask then is, Did he have a fecal concretion in his appendix?

PATIENT'S PHYSICIAN: He had, or, rather, I should say it had been in his appendix; but the appendix had ruptured at the time of operation, and the calculus lay just outside it, in the peritoneal cavity.

DR. MURPHY: That statement clears up the matter. Perhaps some of those present may remember that in the second article which I wrote on appendicitis, in 1890, I called attention to the fact that the early symptoms of an acute appendicitis may be those of an acute peritonitis when a concretion leads to an ulcer at its site and subsequent perforation and leakage. Then the stormy onset of an acute peritonitis is the first intimation that anything is wrong with the appendix. In this case not only did the calculus produce an ulcer and a seepage into the peritoneum, but it produced an area of pressure necrosis so large that when a little infection was added to it, the stone popped out into the peritoneal cavity. How old is the patient?

INTERN: Thirty-seven years.

DR. MURPHY: Was a drainage-tube put in at the time of the primary operation?

INTERN: Yes.

DR. MURPHY: Was the tube removed after eight days?

INTERN: No. The patient states that it was eight days after the primary operation when he first noticed fecal matter draining from the wound.

DR. MURPHY: Had the drainage-tube been removed before the abscess formed two months later?

INTERN: I do not know.

DR. MURPHY: But you *should* know. Had the tube been removed and had all discharge apparently ceased before the appearance of the abscess? If this was the case, and later an abscess and a second fistula formed spontaneously, we should have to think seriously of the possibility of tuberculosis or malignancy in the ileocecal region, since they often begin under the guise of an acute appendicitis, and only later, by their course, reveal their true nature.

PATIENT'S PHYSICIAN: The drainage-tube was removed seven days after the original operation and remained out; but pus kept running continually from the drainage opening during the two months following operation, and practically ever since.

DR. MURPHY: The presence of the fecal concretion was evidently responsible for the early development of peritonitis and for the spot of gangrene in the cecum from which, probably, the greater part or all of the present fecal flow is coming.

One has here a very serious situation to deal with in operating in the presence of such an infection; first, because the feces are running out over the field from both fistulous openings; second, because there must be very extensive adhesions present, and we do not know how many perforations of the intestines around that primary pocket of infection there are. Some of you may remember the similar case we operated the other day where the holes in the bowel were so many that we stopped counting them and we finally had to take out a section of the bowel because it was so riddled with holes, and make an end-to-side anastomosis of the ileum to the ascending colon.

[Dr. Murphy then followed his usual operative procedure, which has been described elsewhere in full.]

Let the record show that at the previous operation for relief of the fistulas a side-to-side anastomosis of ileum to ileum was made; that we found the anastomosis opening patent, but that the coil of intestine above the anastomosis was distended, whereas the intestine below the anastomosis was empty and collapsed; that there were three separate openings from the bowel into the fistulous tract—one in the caput coli, about three-fourths of an inch in diameter, and one in each end of the anastomosed loops of ileum. Let the record further show that we resected the bowel involved in the previous anastomosis and performed an end-to-side union of the ileum, the proximal end being implanted into the distal side; that the intestinal anastomosis was made with two rows of linen sutures; that the opening in the cecum was closed with mattress Czerny stitches of Pagenstecher linen covered with a Lembert suture of catgut; that the blood loss was small, and that most of the operation and all the separation of adhesions were carried out extraperitoneally; that the operation lasted two hours and twenty minutes; that a small strip of gauze was permitted to remain. The patient will be put in a semi-sitting position immediately upon his return to bed, and will also be given an anodyne. Note that we carefully inspected the field to see that no sponges or instruments remained. This is the kind of a case where, unless one is very careful, something might be left in the wound.

The four other cases of fecal fistula which we have operated during the last three weeks have all healed up nicely. We hope that this patient will do as well.

[Note.—He did—primary healing! See the accompanying letter from him, which appears just as it came, without editing.—Ed.]

——, Iowa, 12–16–14.

[Copy.]

Dr. J. B. Murphy, Chicago, Ill.

Dear Doctor: I write you in regard to my condition. I am feeling fine, gained 45 pounds since I left the hospital August 1st. What would be your advice as to my going to work? I am still wearing an elastic bandage, eating good and in a general way feel good. I do not think that I am extra strong yet but gaining in weight fast—weighing now 165.

Kindly advise me by return mail what I had best do.

Respectfully yours,

——————.

P. S.—I had a fecal fistula.

INTESTINAL FISTULA FOLLOWING LATE OPERATION FOR APPENDICITIS; MUCH IMPROVEMENT WITH TUBERCULIN INJECTIONS.—OPERATION; RECOVERY

HISTORY

THE patient, a male twenty-four years of age, entered the hospital November 24, 1914. His family history is negative as to tuberculosis. He has had the usual diseases of childhood. He went through an attack of typhoid in 1907, was in the hospital three weeks, and was sick in bed altogether six weeks, without complications of any sort. He had scarlet fever ten years ago (in 1904) with no complications. He denies all venereal infection.

His present trouble dates back to September, 1913, when a dull ache appeared rather suddenly in the right lower quadrant of the abdomen, with no chills and no fever, so far as he remembers. He did not vomit. The abdomen was rigid, and he was tender over the right lower quadrant. This condition lasted four days, at the end of which time he was operated for appendicitis. He does not know whether or not the appendix was taken out. A gauze drain was inserted, which remained for four days and was then removed. There was no marked drainage after the removal of the gauze until the tenth day of the illness, when he developed severe pains in the abdomen, much distention, fever, vomiting of material like intestinal contents, but no chill. He had had no bowel movement for four days prior to the onset of the pains. This condition lasted for two days. At the end of this time the wound discharged a black, offensive material from the site of the previous drainage opening. Seven weeks after the operation the wound gaped at another spot and discharged some purulent fluid. The pus has continued to discharge from the second opening up to the present time. The lower opening discharges a dark, offensive material, and the upper opening pus. He was given a Koch tuberculin test, and had a pronounced reaction—a temperature of 102° F. on the next afternoon. He was then put on tuberculin treatment, and has gained 35 pounds in weight since it was instituted, about seven months ago.

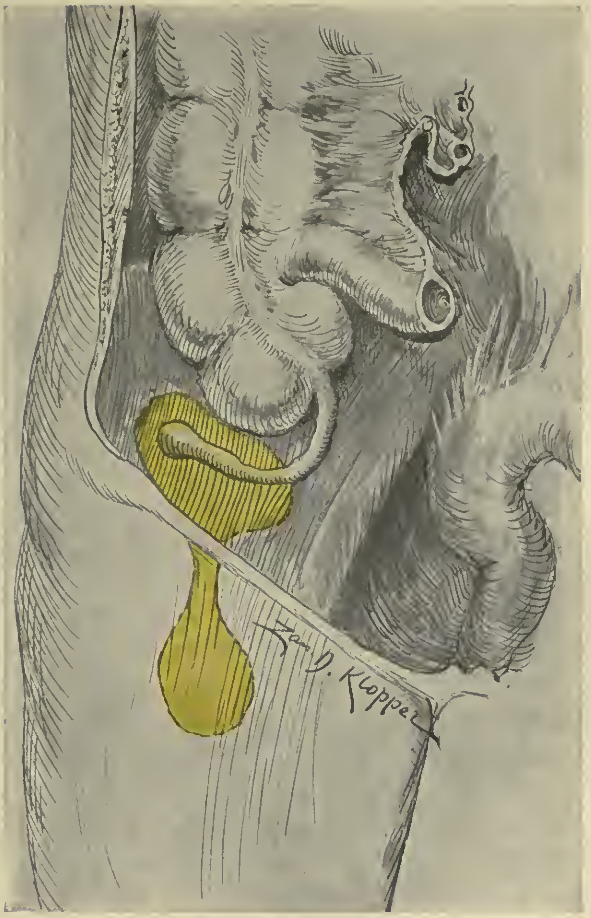


Fig. 7.—Tuberculosis of the appendix with a cold abscess forming and burrowing along the anterior surface of the iliacus muscle under Poupart's ligament into Scarpa's triangle. The abscess in an ordinary "pus appendix" practically never follows this route (from Dr. Murphy's article in Keen's "Surgery").

COMMENTS AND OPERATION

Dr. Murphy (November 25, 1914): In what condition was the appendix found?

Intern: I do not know.

Dr. Murphy: But you *should* have found out something about it. The patient knows about it because he told me. See how important it is in this case to know what the operator did! The patient states that he was first told that the appendix was removed, but in a subsequent conversation the surgeon said he did not remove it. If the appendix, therefore, is still inside the patient, it ought, in all probability, to come out, since, even if it is not the direct cause of this prolonged suppuration, it is very likely to suffer a fresh and dangerous flare-up at any time.

It is also not at all impossible that the patient had originally some focus of intestinal tuberculosis in or near his cecum. The zone of the ileocecal junction and the appendix are the most common seats of intestinal tuberculosis. A point which still further increases the probability of tuberculosis is the fact that we made a tuberculin test and secured a very pronounced reaction. Furthermore, the patient has been on tuberculin treatment since April and has gained 35 pounds in that time. A tuberculous intestinal fistula is one of the most disagreeable conditions in the abdomen to handle, because when it is cured it doesn't stay healed. That sounds like a Celticism, but it is a fact. One may succeed in closing such a fistula, and just as one is congratulating himself on having produced a cure, it breaks out again. If this fistula should prove to be of tuberculous origin, the outlook should be much above that of the average case, because of the constitutional improvement which has taken place under the treatment by tuberculin. Therefore we have decided to make an attempt at a radical operative cure at this apparently auspicious time.

[Dr. Murphy followed his usual technic, which has been previously described.]

There is nothing about the pathologic findings in the case that makes it look typically tuberculous. We have found only one communication with the intestine up to date. Here is a mass that looks not unlike the tip of the appendix. I do not know that it is for certain. There is the small intestine. I thought at first it was the large intestine. I am rather happy about it, because that means we are near the end of our troubles, and this has been a difficult case. Now we shall put in the tube.

Let the record show that there were enormous adhesions in all directions; that the sound introduced as a guide led down a sinus that penetrated in the direction of the spine; that the appendix was found embedded in the mass of adhesions, was greatly thickened and infiltrated, but showed no evident tubercles or foci of caseation; that there were two holes in the intestine—one large one in the caput coli, which was so fixed that it was impossible to bring it up out of the wound, so that it had to be closed *in situ*, and another smaller one in the small intestine, which was not fixed and was easily brought out of the wound and sutured.

We shall have the appendix examined microscopically to see whether or not it shows evidence of tuberculosis. It is enlarged to the diameter of my middle finger and densely infiltrated throughout its entire course. Its proximal end was occluded. We inserted a single small rubber tube for drainage.

The patient will be given nothing by mouth for five days.

December 8, 1914: Let the record show that we shortened the tube one inch, since there has been no fecal discharge; but we retied the silkworm suture, retaining it so as to take no chances on the escape of the tube.

December 15, 1914: The drainage-tube was removed altogether, since there is practically no discharge.

[Note.—The patient made an uneventful recovery.—Ed.]

[Pathologist's Report (Dr. W. O. Sweek).—The appendix shows everywhere a marked round-cell infiltration and considerable lymphatic accumulations. A striking feature of the histologic picture is the enormous number of eosinophiles present. No typical tubercles were seen in any of the microscopic sections, no distinct giant-cells, and no areas of caseation. This histologic evidence would speak for the condition being a chronic inflammatory lesion of the appendix rather than a granuloma.]

THE RELATION OF CANCER RESEARCH TO THE CLINICAL ASPECTS OF CANCER

By Harvey R. Gaylord, M.D.

Dr. Murphy (November 11, 1914): We are very fortunate to-day in having with us a man from Buffalo, N. Y., who is one of the master controlling minds in the research work on the problem of cancer—a man who can give you the up-to-date results of the scientific investigation of that problem in all its relationships. He has been, perhaps, *the* guiding spirit in this work in America. I have the pleasure of presenting to you Dr. Harvey R. Gaylord, of the New York State Institute for the Study of Malignant Disease, to whom we shall be delighted to yield the floor of this amphitheater for as long a time as he is willing to talk to us.

Dr. Gaylord: You undoubtedly know that for the past fifteen years cancer has been the subject of intensive investigation in most of the civilized countries in institutes especially endowed and equipped for this purpose; and there has come into being a great field of activity which is generally spoken of as cancer research. During this period investigators have, for the large part, studied the disease in lower animals, and many interesting discoveries have been made, some of which are calculated to change the classic conceptions of this disease. The time has come when it is desirable and profitable that the general profession should know more of the discoveries of cancer research than has previously been the case.

Our conception of cancer has been greatly modified in recent years. By the propagation of cancer in the lower animals, mostly rats and mice, we have been able to study the clinical course of the disease under experimental conditions, and to establish standards of comparison which are impossible under the conditions which surround clinical observations in human beings. By the artificial propagation of cancers in animals from generation to generation we have been able to give intensive study to neoplasms, a prolonged study of individual tumors which can be carried on over periods of years, instead of the transient study of individual cases as seen in the hospital or dispensary clinic.

The definition of cancer still remains: *A growth springing from the tissues of an individual, composed of cells derived from the normal preëxisting cells which have, however, been so changed as to make them cancer-cells.* These changes endow the cell with the power of limitless proliferation at the expense of the surrounding tissues. The power of growth may be such that when they are transplanted to distant regions of the body they may, on finding suitable environment, produce secondary growths or metastases. This, you see, is still a very broad and a very crude definition, and cancer research has finally taught us, through the intensive study of different neoplasms in the same or different animals, that each growth possesses peculiarities of its own as to behavior or in the possession of the different attributes above enumerated. These propagated tumors may, for a long period, fail to exhibit the phenomenon of metastasis formation, but at any time this "malignant" characteristic may appear. The speed of growth of the cells and the other characteristics of such growths which we describe by the inadequate term "malignant" may vary in a given tumor at different periods. Thus a tumor which in the beginning grows but slowly in inoculated animals may ultimately acquire a very high degree of activity or "malignancy." This, again, it may lose, and the factor of malignancy may be increased or decreased by experimental manipulation.

One of the difficulties in the study of cancer is that without doubt our conception of what constitutes a cancer is too broad. Any growth of tissue which presents the characteristics included in our definition is called a cancer. Cancer research has progressed to the point where we know that, from the standpoint of etiology, there is the greatest possible difference between, let us say, a chicken sarcoma and a sarcoma of the rat. Through the publications of Peyton Rous we have come to recognize a group of sarcomas in chickens which present all the characteristics of genuine sarcoma. By that qualification I mean that they fulfil all the requirements of laboratory manipulation and study demanded of a transplantable sarcoma, as well as presenting under the microscope the characteristic morphologic picture of sarcoma. Rous has shown for three distinct types of chicken sarcoma that they are caused by a virus which may be filtered from the ground-up sarcoma cells through a germ-proof porcelain filter. This virus may also be obtained from dried sarcoma cells, and it will keep when the dried cells are glycerinated, as do other viruses, for long periods, probably years, and still retain its full activity. For many years research laboratories have had transplantable sarcomas in the rat which permitted a prolonged and intensive study. These rat sarcomas present a picture under experimentation differing in no essential particular from the chicken sarcomas of Rous; and yet no one has ever been able to filter a virus from the rat sarcomas.

Thus we see that even between the varieties of relatively so simple a neoplasm as sarcoma there exist very fundamental differences from the standpoint of etiology. Furthermore, the three chicken sarcomas vary in their morphologic characteristics, one being a simple spindle-cell sarcoma, another a spindle-cell sarcoma with a peculiar mode of growth, and the third an osteochondrosarcoma. The virus of each type when injected into susceptible chickens produces only a sarcoma of the exact type and character of the sarcoma from which the virus was taken.

So to-day it is clear that the group of diseases which we call neoplasms is made up of many different varieties when considered from an etiologic standpoint. Morphologic differences have been thoroughly studied, but the study of etiologic differences opens up a new field of work. For this reason we find it to-day profitable and necessary to study cancers individually rather than to attempt to solve the problems of cancer by making generalizations applicable to a whole group. We are now in about the same situation in cancer study as were the medical scientists of two generations ago in the matter of abscesses. At that time it was proper simply to speak of an abscess; but to-day the bald diagnostic statement that a patient has an abscess conveys insufficient information. What one also desires to know now is what organism is the cause of the particular abscess. The demand of the future in cancer research is that we shall know not only the morphologic type of the neoplasm, but also its cause. From this viewpoint we may discover that certain growths, such as dermoids, which have hitherto been included in the group of neoplasms, have but slight, if any, relation to the malignant growths which destroy the affected individual and which we call cancer.

So by prolonged study and experiment we have come to know something fairly conclusive about a certain number of neoplasms in the lower animals. The chicken sarcomas are known to be infectious and are caused by a filterable virus. There is a definite type of round-cell sarcoma in dogs which also is probably of infectious origin. There is a type of sarcoma in the German hare quite likely infectious in nature. There are true epitheliomas in the esophagus and stomach of the rat, experimentally produced by Fibiger, which result from feeding to rats the bodies of cockroaches which contain a minute nematode. These tiny worms penetrate the wall of the esophagus or stomach and in a certain proportion of cases epithelioma develops. The embryos of these worms pass through the intestinal tract of the rats; and if the feces of such infected rats are fed to cockroaches, the nematode embryos develop in the muscles of the cockroaches and the infected cockroaches may be used to infect another series of healthy rats. Whether the worms are the direct cause of the epithelial proliferation, in which case it is assumed that toxic waste products which they elaborate stimulate the epithelium to increased growth, or whether the cockroaches merely act as carriers for viruses such as Rous has discovered in the chicken sarcomas, is not yet determined.

There is a type of growth in certain fish which is a border-line neoplasm affecting the thyroid. It presents early the characteristics of endemic goiter, and in the last stages the characteristics of cancer of the thyroid. This same disease has been produced in dogs by feeding them the scrapings from troughs in which fish have acquired the disease; and in the thyroid of the infected dogs a hitherto unknown minute nematode has been discovered. Endemic goiter has been produced in Europe by administering to rats and dogs the water from wells known to produce goiter in man; and here Bircher has shown that the agent probably passes through a germ-proof filter. McCarrison, in India, the observers in Switzerland, and we ourselves have shown that boiling the water under these conditions destroys the agent of the disease. From these facts one cannot but be convinced that there are several neoplasms in the lower animals of probably infectious origin. It is also quite clear that cancer is not an etiologic entity, but that in the cancer group there are many distinct tumors arising from specific causes.

One of the most important contributions to our knowledge of cancer, as the result of cancer research, is the discovery of an immunity to the disease. Immunity was first definitely demonstrated in mouse cancer by the observation that mice which had been successfully inoculated with the growth frequently recovered spontaneously, and that once they had recovered they could not again be successfully reinoculated with the same tumor. Furthermore, it was demonstrated that the blood of recovered animals had a slight immunizing or curative effect when injected into inoculated animals in the early stages of the disease. This fact indicated the production of a passive immunity, and while first denied by many observers, has now been sufficiently confirmed by various experimenters and with different neoplasms to establish its validity. Clowes, in 1904, showed that if the blood of recovered mice was admixed with a given proportion of cancer tissue to be used for inoculation, in many cases no growth resulted, thus showing that the blood of recovered animals contains antibodies capable of acting upon the cancer-cells in such a way as to deprive them of a cancer characteristic, the possibility of transplantation. This last observation has recently been confirmed by Bashford, who has shown that when cancer-cells are injected into the circulation of resistant or recovered animals, they do not grow.

Goldmann, of Freiburg, in the nineties, made a careful study of cancer of the breast and showed by microscopic sections that cancer-cells penetrate the vessel-walls and are free to be transported in the circulation from the very beginning of the growth.

He reasoned that if cells find their way into the circulation in the early stages of the disease, but are unable to produce metastases, there exists an immunity to the cancer-cell, probably active and present in the blood, which protects the individual until such time as the immunity fails in the later stages of the disease.

Thus cancer presents itself to us as a disease in which, through the action of some unknown cause or causes, the tissues are stimulated to abnormal growth. This growth and the clinical course of the disease are, however, modified by the development of or the preëxistence of certain immune substances in the individual. It has been shown by experiments on animals inoculated with cancer that if in the early stages of the disease a part of the tumor is removed, or hemorrhages are induced in it by manipulation or by the injection of such substances as adrenalin, the tumor will frequently retrogress and disappear, whereas the same procedures in the late stages of the disease simply encourage the tumor to more rapid growth. This phenomenon is due to an immunity which arises in the course of the development of the neoplasm.

By a careful study of spontaneous recovery in mouse cancer we have been able to show that the chances of such recovery are greatest in the early stages of the disease and decrease with the progress of the disease. Therefore the chances of success of surgical interference, or of any other manipulation well calculated to destroy a cancer, stand in direct relation to the possibilities of early application. The reasons for sending cancer cases to the surgeon for early operation are based not alone upon the possibility of removing the entire growth, but upon the fact that in the early stages of the disease the processes of immunity are still active. All surgeons of experience have operated upon early cases of cancer where, although they have felt morally certain that they were able to remove the entire growth, no recurrence took place and the patient remained well. Furthermore, in the late stages of cancer, when immunity has practically disappeared, surgeons have frequently noted that operation seemed simply to make the condition worse.

We have as yet no means of determining positively whether the immunity in a given case is present or has failed. With inoculated animals it is possible to show the existence of a concomitant immunity by attempting secondary inoculations. A mouse which has been inoculated and has developed a tumor of moderate size, as a rule cannot be reinoculated in another locality. In the last stages of the disease, however, reinoculation may become possible.

When we see a patient who has remained in good condition, the growth fairly stationary, for a long period and with no metastases, we must infer that such a patient possesses some immunity to the disease. In the Buffalo laboratory years ago we were able to show that when a tumor successfully inoculated into a mouse tends to remain stationary for a considerable period of time, repeated anesthesia with ether or chloroform over a period of several days will often cause the tumor to grow rapidly. This rapid growth, we must infer, is due to injury to the immunity-producing mechanism. Whether this injury is due to the destruction of antibodies in the blood or to damage of the organs which produce the immunity we do not know; but it suggests to us the possible desirability of limiting the amount of anesthetic used in operations for cancer. Possibly it would be wise to operate cancer cases with gas when possible. Experiments have also shown that prolonged anesthesia has an unfavorable effect upon the course of ordinary bacterial infections (Rubin, Evarts Graham).

We must naturally ask ourselves what is the nature of cancer immunity and how is it produced? For some years cancer investigators have known that a certain amount of resistance to implanted cancer could be induced in susceptible animals by inoculating them with normal tissues. Various tissues, embryonic and adult, and even the blood, are capable of producing this type of mild immunity; but the best of all tissues used for this purpose has proved to be the spleen. Investigators found that by removing the spleen, animals can be made more susceptible to inoculations with cancer. Within the last year or two Dr. Murphy, of the Rockefeller Institute, has shown that various neoplasms can be inoculated with great facility into the partly developed embryo of the chick. The first experiments of this kind were made by Rous, who inoculated his chicken sarcoma into partly incubated eggs. Then Murphy showed that rat sarcoma and mouse carcinoma can be grown in chicks, although it is practically impossible to grow a mouse carcinoma or a rat sarcoma in an adult animal of a different species. Murphy then found that if one implants in a chick embryo some adult splenic tissue with the neoplasm, whenever the splenic tissue grows the neoplasm does not. He further extended this research and showed that when a neoplasm has started to grow in a chick embryo, if adult spleen is inoculated and grows the neoplasm will retrogress. From this observation he concluded that the growth of the spleen is antagonistic to the growth of certain neoplasms.

Various observers have shown that the removal of the spleen from a normal animal increases its susceptibility to inoculation, and Murphy obtained similar results by treating mice or rats with repeated daily doses of the *x*-ray for the purpose of injuring the lymphatic apparatus. By so doing he has been able to grow mouse and rat cancer in animals of alien species, the tumors growing well until such time as the lymphatic apparatus recovers from the *x*-ray injury. Then the tumors retrogress. He has also shown that if the lymphatic apparatus is thus injured, animals which ordinarily are not susceptible to infection with tubercle bacilli become susceptible.

It thus appears that the immunity to neoplasms, as also to bacterial infections, and even to alien normal tissues, is produced by the lymphatic apparatus, the principal organs of which are the spleen and bone-marrow. I have had placed upon the blackboard a list of 516 primary neoplasms. There are 156 of the stomach, 95 of the female genitalia, 57 of the esophagus, 55 of the intestines, 30 of the mamma, 19 of the gall-bladder, 18 of the prostate, 17 of the lung, 8 of the pancreas, 8 of the skin, 5 of the urinary bladder, 5 of the liver, 5 of the tongue, 3 of the branchial clefts, 3 of the larynx, and 2 of the upper jaw. They were taken from a series of autopsies performed in a great Continental hospital. One sees at once that the list does not contain a single case of tumor of the spleen or of the bone-marrow. In fact, there are only about 30 credible cases of so-called sarcoma of the spleen. The primary malignant tumors of the bone-marrow are also extremely rare and present a curious border-line picture, the neoplasms being known as chloroma and myeloma.

Metastases of malignant tumors in the spleen or bone-marrow are also rather rare and occur late in the disease. In the list on the blackboard they represent only 2 per cent. of all metastases, and this I believe to be an unusually high percentage. This great immunity of the spleen and bone-marrow to the development of primary tumors, as well as the great resistance of these organs to secondary growths, is easily understood in the light of our knowledge of the production of immunity to cancer and of our understanding of these organs as the principal sites of the development of immunity in general. It is needless to state that the understanding of these facts makes it possible for us also to understand certain vagaries with regard to cancer cases. They suggest, too, that the evil results of indiscriminate roentgenotherapy in human beings in the treatment of cancer may in some cases be due to the injury to the lymphatic apparatus thereby brought about, and the consequent diminution of the immunity. This theory will also explain those remarkable but rather frequent cancer cases where apparently marked improvement after one or two treatments with the *x*-ray or radium is quickly followed by rapid return and extensive metastases when the *x*-ray or radium treatments are carried further.

Susceptibility to cancer, it has now been shown by Dr. Maud Slye and other investigators, may be inherited. Dr. Slye has been able to breed families of mice in which most of the animals which have lived to maturity have developed some type of cancer or leukemia. Her results have been used as an argument in favor of the actual inheritability of cancer; but in the light of what is known regarding the production of cancer immunity and regarding the destruction of immunity by injury to the lymphatic apparatus, we need only assume that this inherited quality is a hyper-susceptibility to injury, or possibly an inherited defect in the immune mechanism, and this may possibly be the expression of deficient activity on the part of the lymphatic apparatus.

Dr. Murphy: You have just listened to one of the best discussions of cancer fundamentals that I have ever heard. It was the tremendous amount of information, not details, but salient and important facts bearing directly on the clinical phase of the disease, which makes this talk so important. One of Dr. Gaylord's facts which profoundly impressed me was that many cases of cancer after they reach a certain stage of development are actually injured by operation, that is, life is shortened by the operative procedure. This statement is in consonance with our clinical experience, namely, that late operations for cancer often hasten death. That is the proper way to put it. I have been gradually becoming a pessimist in regard to the operative results in cancer, because in our clinic, operating, as we do, only on referred patients, we see few if any early cases. They are all the late, the difficult, and the hopeless cases. Dr. Gaylord's statements recalled to my mind a remark of the great Benjamin Brodie, made in his latter days: "I should like to have the Lord forgive me for all the deaths I have hastened by operating on late cancer. I believe most of them would have lived longer if I had not operated." In Brodie's time the cancer patients came in a still more advanced stage of the disease to the operating table than they do today. Another statement that impressed me greatly concerned the favorable results obtained in the early-stage operations when the patient possesses an immunity of the highest degree—a natural immunity. This well-grouped observation forces on us an additional obligation to recognize cancer in its earliest stage, if not in the so-called precancerous stage.

These are startling facts to the clinician, but they are convincing. They are all the more valuable because they point out to us so clearly the route we must follow. Cancer is not kind to the surgeon. I have often said that I should like to shift all my cancer cases to somebody toward whom I bear a grudge. It is only the absence of a sufficient personal animus which has kept me from such a step. We are now declining to operate a larger percentage of advanced cases of cancer than we have ever declined formerly.

Another important fact brought out forcibly by Dr. Gaylord was regarding the use of radium and the x-ray, namely, that the case which improves at the beginning of the treatment often grows rapidly worse when the treatment is continued over a considerable length of time, so that the lymphatic apparatus becomes materially disturbed by the rays. That observation corresponds exactly with the statement made to me last summer by Captain French, head of the Radium Institute of London. I asked him quite a few pointed questions about the course of the cases under radium treatment. I asked, among other things, "What about the cures?" He replied, "I assure you, my dear doctor, *we never speak of cures in adenocarcinoma.* There is only one class of cases that is cured with radium and remains cured, and that is the superficial epithelioma of the skin." These basal-cell epitheliomas recover also with the x-ray and with "pastes." The number of cures has not been materially increased by the use of radium and the x-ray. Whether the life of the average patient has been materially prolonged remains an open question. We know positively that cases of both sarcoma and carcinoma which have been running a relatively benign course so far as the patient's comfort in life is concerned, when disturbed in an operative way or by x-ray treatments seem to undergo a malignant metamorphosis and rapidly and cruelly terminate life.

I can cite a case which occurred in the last week of my service at the College of Physicians and Surgeons in Chicago, about sixteen or seventeen years ago. The patient was a boy who had a tumor of the kidney which I diagnosed as a sarcoma; and, because at that time there were no recorded permanent cures of true renal sarcoma by removal of one kidney, the growth always recurring ultimately in the other, I said that he should not be operated. In a year I saw him again and the tumor had increased in size. The boy too was growing rapidly. In two years I saw him again. I saw him occasionally up to five years after his first visit. I then concluded that I had made an error in my diagnosis. I decided to operate. I performed a nice, clean-cut separation of the boy from the tumor, because the tumor was about the same size as the boy. In eight weeks he was dead, with a recurrence of the disease on the same side, associated with an enlargement of the opposite kidney, notwithstanding his condition had not seemed seriously threatening at any time during the five years that preceded the operation. The extirpated tumor proved to be a typical renal sarcoma. There is no doubt in my mind, and I think there can be none in yours that the operation precipitated his death.

I am happy that Dr. Gaylord gave us this information. It eases my conscience, which has troubled me on two points. First, if patients of this class live a long time after I have pronounced them incurable and have declined to operate, I often feel that perhaps I should have given them the slight chance which operation afforded; second, when they have died shortly after operation, I have often thought they might have lived longer if I had been more conservative.

The real purpose in putting these cancer statistics (which will be published in an early number of the CLINICS in full) on the board this morning was to call attention particularly to the distribution of the known metastases in carcinoma of the breast. These statistics I do not feel able to accept unreservedly, notwithstanding they were taken from the records of probably the most scientific institution for postmortem pathology that exists to-day—Orth's laboratory at the Charité in Berlin.

You will note in the tabulation of 30 cases of mammary carcinoma that there was only one case in which there was no metastasis demonstrable at the time of death. But let us consider carcinoma of some other organs, and see what percentage of cases had no metastases at the time of death—for instance, the sigmoid, the fundus of the uterus, and the fundus of the gall-bladder. You will find that in the total number of postmortem records of cases of cancer collected and analyzed by me, 10,315, involving all the common sites of cancer in the body, 19.7 per cent. had no demonstrable metastases whatever at the time of autopsy, meaning that even at death the disease remained local in practically one-fifth of all the cases. Metastases take place less frequently and at a later period when the cancer is situated in the fundus of an organ than when it is in the cervix or canal of exit. This fact is due to a well-recognized condition, namely, the sparsity of lymphatics in the fundus of most organs and the richness of the supply in the cervical zones. We have always believed that the relative richness of the lymphatic supply plays an important rôle in the time of spread of cancer and in the frequency of cancer transmission to distant organs. We find in Handley's work that he too has accentuated the statement that the relative richness in lymph-spaces and channels is an important and determining factor in the time and frequency of transmission of carcinoma to neighboring structures.

Another topic which I hope Dr. Gaylord will allude to tonight (in his paper before the Chicago Medical Society), because it is one that interests us very much, concerns the sites of election of metastases from primary cancer in the various organs. Why does cancer in one organ form metastases in certain positions and why not in others? The question seems to be intimately bound up with that of embolism in general. You may remember that Stephen Paget, in speaking of embolism, stated, on *a priori* grounds, that the frequency of embolism should be equal in all tissues and organs. We know that such is not the actual fact. One never sees an embolus in a tendon, for instance. We know that although emboli of various sorts may be passing through the circulation of all the various structures, they lodge, develop, and produce symptoms only in certain elective localities; but what conditions control their arrest and growth in one tissue and their apparent freedom from arrest in another we do not yet know. This is a matter of vital interest to us at the present time, and we are very anxious to have it cleared up, if possible.

I wish again to thank Dr. Gaylord for his kindness in presenting this important subject in my clinic.

ANEURYSM OF THE BRACHIAL ARTERY—ENDOANEURYSMORRHAPHY

HISTORY

The patient is a married man, aged forty-three years, a physician. His family history is negative. He had the usual diseases of childhood, including measles. He has had a number of attacks of tonsillitis—two or three a year, as a rule. He had acute articular rheumatism in 1905, involving the shoulder-, the wrist-, and the elbow-joint on the right side. He was in bed for one month. Immediately prior to the rheumatic attack he had a severe attack of tonsillitis.

The patient's venereal history is negative. For the past ten years he has been in the habit of drinking a bottle of beer at night before retiring.

Fourteen months ago,—in July, 1913,—while in his berth on a railway train, the patient noticed for the first time a mass on the inner surface of the right arm, two inches distal from the axilla. He palpated it and noticed it pulsating. It was then the size of a pecan. It has not enlarged to any material extent since then. It has never given him any trouble worth mentioning. If there is tight clothing around the arm, he sometimes notices a throbbing. He consulted another doctor regarding this swelling, and the latter advised operation.

The patient has no recollection of any trauma occurring at this site at any time. He has never done any work that necessitated severe exertion. His blood-pressure is now 142 in the right arm and 136 in the left. The tumor is still about the size of a pecan, pulsates, is circumscribed, and lies about two inches distal from the axilla.

COMMENTS AND OPERATION

Dr. Murphy (September 17, 1914): Where did you place the arm-band in taking this patient's blood-pressure?

Intern: Just below the aneurysm when testing him on the right side. As a check I used a second blood-pressure apparatus and secured the same reading as with the first apparatus.

Dr. Murphy: What is the matter with him?

Intern: He has an aneurysm of the brachial artery.

Dr. Murphy: Where?

Intern: About two inches below the margin of the latissimus dorsi.

Dr. Murphy: Just what *is* the matter with him?

Intern: I do not know what can have caused the aneurysm. It came on spontaneously, so far as the patient tells me. He gives no history of any special pain preceding the onset or during the course of the disease. There is no cutaneous scar near the artery to suggest an injury, and there is no history of trauma. His blood-pressure is hardly high enough to suggest it to be the cause of his trouble, and he positively denies syphilitic infection and has no demonstrable evidence of it.

Dr. Murphy: What do you mean by an aneurysm?

Intern: I mean a dilatation of any part of an artery. It may be fusiform or sacculated.

Dr. Murphy: I like your definition, doctor, with the exception of two points: First, an aneurysm is usually, but not always, confined to a *part* of an artery—a cirsoid aneurysm or a dissecting aneurysm may involve an entire artery; second, an aneurysm, while characteristically an arterial lesion, not rarely may occur as a venous lesion or as a combined arterial and venous lesion. According to its Greek etymology, the word aneurysm means simply a dilatation; but by common consent and usage we have limited the term to pulsating vascular dilatations. If pulsating dilatations ever developed in connection with the lymphatics, I believe that we should have to call them aneurysms. How do you classify aneurysms?

Intern: As spontaneous and idiopathic.

Dr. Murphy: No. I do not like your classification. I think that perhaps the most logical classification is into congenital and acquired forms. Since the congenital variety is so rare, however, that it constitutes less than $\frac{1}{10}$ of 1 per cent. of the cases, a more practical and more usual classification considers only the acquired aneurysms, and divides them into the true and the false or spurious forms.

While this is the classification found in most pathology text-books, it is made essentially from the standpoint of the morbid anatomist. For the purposes of the surgeon and therapeutist, a classification on an etiologic basis is more useful than one from an anatomicopathologic point of view. Usually I feel that it is of less importance in a given case to determine whether the aneurysm is true, false, or dissecting, sacculated or fusiform, than whether it is traumatic, embolic, erosive, inflammatory, luetic, arteriosclerotic, or "idiopathic." I use the last term not because it signifies anything more than etiologic ignorance, but because it represents a group of cases, in operating on which one must be prepared to cope with any possible etiologic factor, no matter how bizarre.

Let us first consider the aneurysms of traumatic origin. The trauma may be a contusion, a laceration, a fracture, or a bullet wound. The most common cause of traumatic aneurysms probably is a bullet wound, the next a stab-wound, and the next a contusion.

Aneurysms of traumatic origin are not infrequently of the dissecting type. Dissecting aneurysms may present just as disagreeable surprises as do those of the "idiopathic" type. A dissecting aneurysm may be very misleading in its symptomatology because it may develop as a non-pulsating tumor, the rate of growth of which presents great variations; and, furthermore, the tumor may appear clinically at a considerable distance from the site of rupture of the arterial wall.

I recall a case from the practice of Dr. Edward Lee in 1879 which made a great impression on me at the time. The patient had been shot in the face with a 22-caliber bullet. He first consulted Dr. Lee in order to have the wound examined and dressed. The bullet had entered the right cheek somewhat below the orbital border, and was believed to have passed downward, backward, and outward in its course. It was impossible at that time to state just where the bullet had lodged because the patient had no focal symptoms referable to it and lived twenty years before the *x*-ray era. No probing was done by Dr. Lee, but a dressing was applied and the wound in the face healed in a few days.

Six weeks later the patient, accompanied by a friend, returned to Dr. Lee's office complaining of a painful swelling which was developing in the roof of his mouth. Dr. Lee on examination found the swelling located above and to the outer side of the right tonsil, displacing the velum forward and toward the midline. It was firm to the touch, very tender, and non-pulsating. Dr. Lee presumed that it was a supratonsillar abscess, perhaps originating from the arrested bullet.

I have learned from bitter experience to stick the needle of a hypodermic syringe into every suspicious swelling, whether tense and hard, or soft and fluctuating; but in those preantiseptic days we were always looking for pus and usually found it, and the hypodermic syringe was not in use.

So Dr. Lee incised the swelling. First a few clots of blood slowly wriggled their way out, and, a little faster, a few more. Then came the rush of the arterial current with the full force of the spurting carotid. The patient, strangling in his own blood struggled wildly, and his friend ran away in a panic, an abject deserter. Before Dr. Lee could gain control of the patient the latter had bled to death and the office was like a shambles from the struggle

Dr. Fenger performed the coroner's autopsy. He demonstrated that the bullet had passed through the anterior wall of the internal carotid and lodged with its base projecting into the posterior wall. From there a dissecting aneurysm had started between the adventitia and media and had burrowed upward and forward to its position above the right tonsil.

Trauma occasionally produces arteriovenous aneurysms also; but they are rather rare. I had an interesting case of this kind in 1892. The boy had been shot in the neck some months before he came to me for treatment. On examination I heard a loud buzzing bruit at the base of the neck on the right side. The tissues of the neck were but slightly elevated, and there was no great dilatation of the cervical veins; but the external jugular

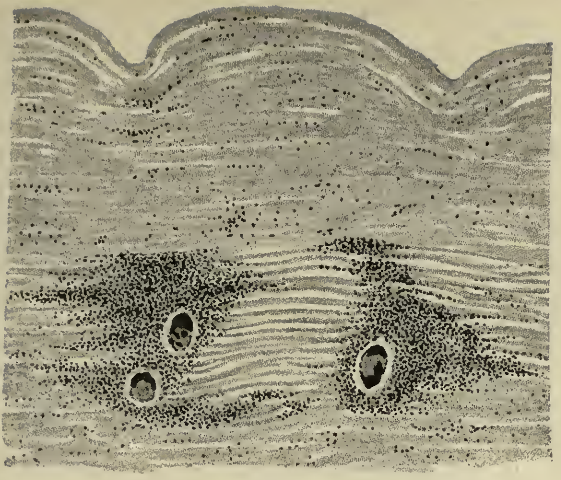


Fig. 8.—Gummatous aortitis (mesaortitis) (microscopic appearance, partly schematic). The intima is thickened, sclerotic, and thrown into fine folds. The media contains infiltrates, especially around the vasa vasorum, consisting largely of plasma cells with a few giant-cells. Such a small gumma, or series of gummata, heals with granulation tissue and a thin scar, leaving a weak spot in the media which readily stretches and gives way. Secondary changes in the intima, atheroma, and calcification are very frequent sequelæ of gummatous mesaortitis.

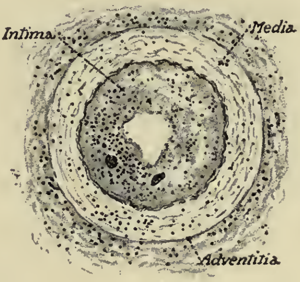


Fig. 9.—Tuberculous arteritis (microscopic and largely schematic). Note the round-cells in the adventitia, the partly infiltrated and partly hyaline media, and the proliferating intima containing tubercles, giant-cells, and some foci of caseation. Such a process may lead to the formation of small aneurysms, as shown in Fig. 11, No. 8.

vein pulsated synchronously with the carotid. The patient's greatest complaint was the noise which the bruit of the aneurysm produced in his ears, and this noise could be heard by placing one's ear against the patient's ear.

In operating I exposed the carotid and internal jugular and located the point of anastomosis. A double ligature was thrown around the connecting isthmus without division of the latter. This simple ligation shut off communication and resulted in a complete and permanent cure.

A certain number of these etiologic varieties are commonly grouped under the name of "spontaneous" aneurysms. What are they?

INTERN: Those due to high blood-pressure, syphilis, and arteriosclerosis.

DR. MURPHY: Better say those due to pathologic changes in the arterial wall. The most common cause is syphilis; the second, endarteritis. By that statement I do not mean to imply that endarteritis is never of syphilitic origin. No doubt frequently it is. But mesarteritis is the characteristic syphilitic change, particularly in the larger vessels, and it is with the larger vessels that we have to deal, as a rule, in aneurysm. Both mesarteritis and endarteritis weaken the vessel-wall by producing a scar in it. This scar may then stretch and thin out, or it may undergo calcification, with subsequent fracture of the calcified plaque and entrance of the blood into the wall of the vessel. What other causes are there?

INTERN: High blood-pressure.

DR. MURPHY: Yes, that is frequently an associated condition. But I refer rather to other direct causes. Some that are well worth mentioning are embolism, both bland and mycotic, erosion of the vessel-wall by tumors, suppuration, tuberculosis, and other granulomata, and by parasites, the *Strongylus armatus* being not infrequently the cause of aneurysm in the mesenteric arteries of horses. This man has none of these etiologic conditions.

Let us next consider the anatomic forms of aneurysm: Sacculated, fusiform, complete and incomplete, and cirsoid. Let us commence with the last mentioned. What do you mean by a cirsoid aneurysm?

INTERN: An elongated aneurysm.

DR. MURPHY: It is an elongated, worm-shaped aneurysm, consisting of a diffuse dilatation of one or more arteries, involving large stretches of the vessel, but attaining no marked focal enlargement at any point. It resembles in the arterial wall the change which one sees in certain progressive varicosities of veins. These cirsoid aneurysms occur most commonly in the hand, head, elbow, and ankle. Cirsoid aneurysms of the hand are extremely difficult to treat successfully. In cirsoid aneurysm of the scalp, when one carotid is involved in the growth the affair is always serious. When the two carotids are involved, it makes a difficult and terrifically dangerous condition to treat by surgical or other measures.

I well remember the first case of extensive cirsoid aneurysm which I saw. It was a patient of Dr. Lee's, a cranial cirsoid aneurysm, in which Dr. Lee performed a double ligation of the external carotids. The patient died of sepsis. The postmortem examination showed that the result of the ligation was the production of a septic thrombosis. That was just the beginning of the antiseptic era. A dissection of a cirsoid aneurysm is very much like the dissection of those progressive varicosities that sometimes occur in the arm, as in a case I had recently. Excise one set of vessels, and the growth spreads to others. Excise those others, and still further groups become involved. From one vascular zone it spreads to another.

I recall seeing a case last year in Detroit, a cirsoid aneurysm of the palmar arch, operated and reoperated, ligated and religated, dissected and redissected, and still a collateral circulation always developed and the growth recurred. That is usually what happens with the cirsoid type of growth in any position in the body. By some pathologists—among them Virchow, who did the first important work on this lesion—it is actually considered a tumor, and therefore called *angioma arteriale racemosum*. It is a lesion which, when occurring in an arterial zone, resembles a malignancy in its behavior. Cirsoid varicosities in the hand one may repeatedly excise; but they usually recur and always tend to a fatal termination. Fifteen, twenty, twenty-five, and thirty years may elapse from the time the growth starts in the hand until the varicosity extends into the mediastinum, and the patient commences to suffer from the pressure effects on the respiratory organs. Then it terminates fatally. When one grasps the hand in these types of varicosities it feels like a mass of bones covered with skin and padded like a feather pillow; but, nevertheless, muscular action is never lost. Some years ago there was a man who made his living showing himself around Chicago as "the human skeleton." He had a cirsoid type of varicosity.

Of the aneurysms occurring in the extremities, the most common is that of the popliteal artery, usually due, at least indirectly, to the trauma and stretching which occur in the popliteal space in persons who use the legs excessively. It represents from 35 to 50 per cent. of all aneurysms of the extremities, and exceeds in frequency of occurrence aneurysms in all other positions except the aorta.

Popliteal aneurysms due to a single injury are infrequent. With few exceptions they arise spontaneously, either on a syphilitic or atheromatous base; but given this *locus minoris resistentiæ*, the pulling stresses in the popliteal region hasten the rapid development of the lesion. The pulsating tumor soon presses on the popliteal veins and nerves. Cyanosis and excruciating neuralgias set in in the ischemic leg, and the aneurysm rapidly burrows to the surface and becomes adherent to the skin. Then it may burst to the outside or rupture beneath the fascia. In the latter event it forms an enormous spreading hematoma, the pressure of which soon stops all the circulation below the knee and produces gangrene. Under rare circumstances an occasional aneurysm may burrow its way forward instead of backward and rupture into the knee-joint or erode by its pressure one of the condyles of the femur.

Popliteal aneurysms are not infrequently mistaken for abscesses or cysts, particularly posterior bursal hygromata, and this error is all the easier to fall into because not all popliteal aneurysms pulsate. Those which have a narrow communication with

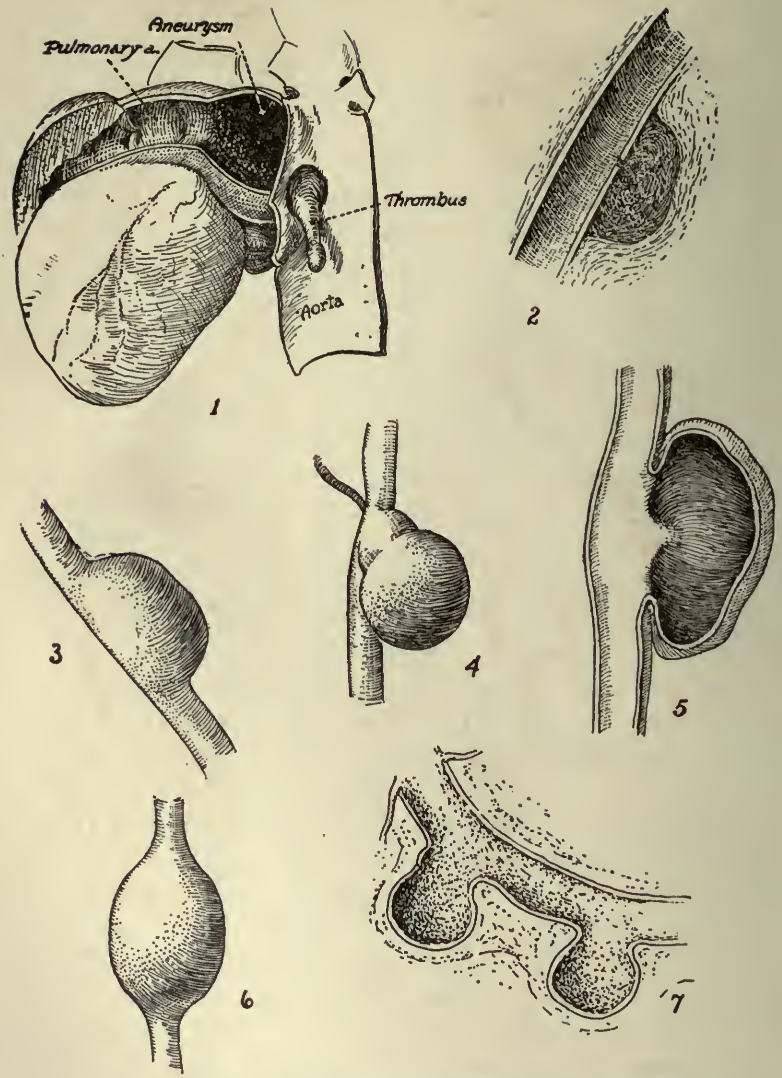


Fig. 10.—Types of aneurysm (largely schematic, after Kaufmann).

1, Congenital aneurysm of the ductus arteriosus (Botalli), filled partly with a thrombus which projects from the aneurysm into the aorta. The patient was an eleven-day-old female infant who died of sepsis starting in an umbilical phlebitis.

2, False aneurysm due to perforation of the vessel-wall. Note that the aneurysm lies entirely outside the vessel, and is bounded by the perivascular tissues only.

3, Scaphoid type of true aneurysm.

4, Sacculated aneurysm of the basilar artery (slightly enlarged over natural size).

5, Sacculated aortic aneurysm divided in half longitudinally (much reduced in the reproduction).

6, Fusiform aneurysm.

7, Miliary aneurysms of a cerebral artery (in cross-section, magnified).

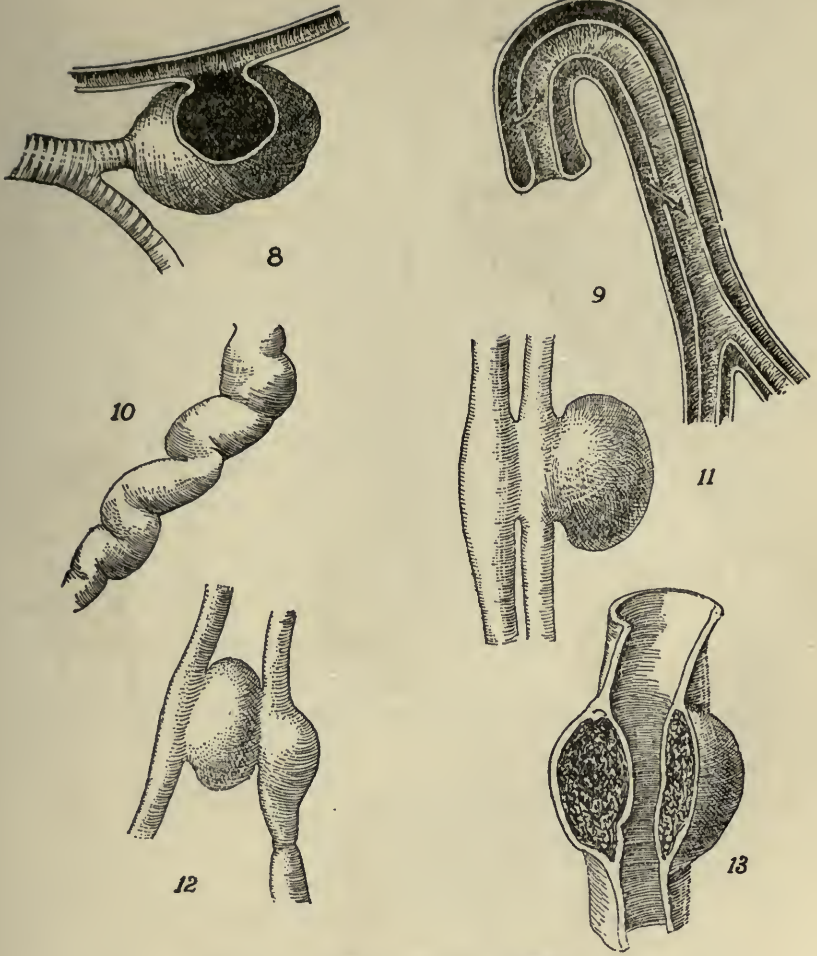


Fig. 11.—Types of aneurysm (*continued*).

8, Aneurysm of the pulmonary artery projecting into a small tuberculous cavity, the latter connected with a bronchus. This is one type of vascular change which may produce fatal hemoptysis in pulmonary tuberculosis. (See Fig. 9, for the microscopic picture of early changes in such a vessel—tuberculous arteritis.)

9, Diffuse dissecting aneurysm involving the entire aorta and both iliac arteries. The arrows indicate the two openings present in this case between the lumen of the aorta and the enormous aneurysmal sac surrounding it.

10, Cirsoid aneurysm.

11, Arteriovenous aneurysm due to a penetrating wound of artery and vein simultaneously. The vein communicated with the artery by a narrow opening, which was large enough, however, to allow enough blood to pass to dilate the vein quite materially. The artery was penetrated at two points by the injury, and communicates through one opening with the vein and through the other with the aneurysm.

12, Arteriovenous aneurysm of traumatic origin. The aneurysmal sac is interposed between artery and vein, and the latter is dilated and tortuous because of the arterial flow into it.

13, Dissecting aneurysm of iliac artery filled with thrombus masses not completely organized. The opening into the sac from the intima cannot be seen in this view.

the artery, or whose wall contains thick layers of organized fibrin, may very closely resemble cystic or solid tumors. *It is a safe rule to regard every swelling in the popliteal space as possibly an aneurysm.*

In the management of these peripheral aneurysms there are as many methods of treatment as there are varieties of aneurysms. The older methods of operative treatment are now chiefly of historic interest, but they were once of great importance to me when I was a student. It was my failure to answer correctly the question on aneurysm which almost cost me my internship in the County Hospital—my first great medical opportunity. The question as put by Dr. Isham was: "Give Hunter's treatment of aneurysm and describe Hunter's canal." Hunter's canal presented no difficulty, but Hunter's aneurysm treatment floored me. The next man knew the answer, and so I have never forgotten it. The treatment was a double ligation, above and below the sac. It was a palliative rather than a curative treatment, but was often successful. We are placing a classification of the treatment, as well as of the pathology of aneurysm, on the blackboard:

CLASSIFICATION OF ANEURYSMS

A. Congenital.
B. Acquired.
    (*a*) False or spurious.
    (*b*) True.
    (*c*) Dissecting.
        1. Spontaneous.
            (*a*) Arteriosclerosis.
            (*b*) Endarteritis.
            (*c*) Mesarteritis (especially syphilis).
        2. Traumatic.
        3. Embolic.
        4. Erosive.
        5. Cirsoid.
        6. Arteriovenous.

The seat of the aneurysm is most frequently the aorta

(75 per cent.); next, the popliteal artery, with 16 per cent. of all cases, or from 55 to 60 per cent. of the peripheral aneurysms.

Males, 78 per cent.; females, 22 per cent.

Usually single; occasionally multiple.

Occurs at all ages. Most common between forty and fifty years (about one-third). About 2 per cent. occur before twenty and 2 per cent. after seventy.

Treatment.—

- Medical.
- Surgical:
  - Refrigeration.
  - Compression.
  - Acupuncture.
  - Filipuncture.
  - Electropuncture.
  - Radical:
    - Aneurysmotomy.
    - Aneurysmectomy.
    - Endoaneurysmorrhaphy.
    - Obliterative.
    - Restorative.
    - Reconstructive.

In the last 85 cases of aneurysmorrhaphy operated by 52 surgeons (49 American and 3 foreign), 77 were typical and 3 atypical. The arteries involved were:

Iliac, 1.
Gluteal, 1.
Iliofemoral, 5.
Femoral, 18.
Popliteal, 50.
Posterior tibial, 1.
Brachial, 2.
Aortic, 2.
External carotid, 1.
Subclavian, 1.
Subclavio-axillary, 2.
Axillary, 1.

Over 50 per cent. of the peripheral aneurysms were popliteal.
Over 21 per cent. were femoral.
Of the 85 operations–
    59 (69 per cent.) were of the obliterative type.
    13 (15.4 per cent.) were of the restorative type.
    13 (15.4 per cent.) were of the reconstructive type.
Of the 85 patients—
    78 recovered.
    7 died.
    4 suffered from gangrene (only in the reconstructive cases).

Relapses occurred only in the reconstructive operation (4 in 13 operations)—28.9 per cent. of the group, or 4 per cent. of the total.

Dr. Murphy: Following the various ligating operations came the excision of the aneurysm, and following excision came the internal ligation of all the vessels found opening into the sac when the sac was opened. The latter was the favorite plan of treatment until Matas' operation came into being in 1908—the suture of the aneurysm wall, endoaneurysmorrhaphy. I think Matas' suture method is better than the plan which I had advocated previously in a paper at the New Orleans meeting of the American Medical Association, namely, the overlapping flap. That method was a sequel of the large amount of experimental surgery on the arteries and veins which I carried out in 1896 and 1897. We had by that time become pretty familiar with vascular work.

In my closing address to the students last year (see Clinics for June, 1914) I stated that no surgeon should start the active practice of surgery until he has done a considerable amount of experimental surgery on the arteries and veins of dogs. The experience thus obtained will be invaluable to him in his later practice, because it adds so greatly to the safety of the patient in his hands. When vascular surgery has been mastered by the prospective surgeon, chest surgery should next be studied on the dog. When this field, too, has been mastered, then the surgeon is pretty competent to undertake abdominal experimental work also, and to do nearly anything that may come along in the experimental line and do it well, and he has a proper foundation laid for the technic of clinical surgery. Of all the interesting experimental work which I have done, there is nothing I look back to with more pleasure than my arterial work.

My method for the management of aneurysm, the overlapping method, is not so good as the endoaneurysmorrhaphy of Matas, and that is why I have now abandoned it altogether and use Matas' method as routine. I have still a few reprints left of my arterial work in 1897, and these I will pass out to you. In it you will find a report of the first end-to-end suture of the artery that was ever accomplished.

We found vascular surgery pleasant and successful work on both the human being and the dog; but we learned the method on the dog. Whenever we work out anything new in surgical technic we always do it on the dog first. Then, after we have made a success of it, we do it on the human being. Acquiring a surgical practice, I think, is only a matter of being ready and equipped to receive it when it comes. When you are ready, the cases are there. The reason the cases do not arrive earlier is that you are not prepared to diagnose and care for them.

The primary cause of the lesion in this case I do not know yet. We have been unable to elicit any definite etiology for the aneurysm in the history or the physical examination, but in view of his frequent attacks of tonsillitis it may well have been a metastatic infection of the vascular wall by way of the vasa vasorum.

In doing this vascular work one has to take practically the same rigid aseptic precautions that one does in bone and joint surgery. Infections here are terrifically serious. There is no field in experimental work where so many surprises occur. For instance, after dividing and suturing the abdominal aorta, when the compress is first taken off the vessel the blood coming through the suture openings will often shoot above one's head. One would think that the dog is going to bleed to death, if one has not seen such cases previously; and if a hemorrhage like that came from a *patient's* aorta, one would certainly put his hands and almost his feet on the vessel in trying to stop the bleeding. If, however, one only keeps cool, presses gently on the vessel with a sponge for fifteen or twenty seconds, or sometimes a little longer, one will usually be surprised how quickly all the hemorrhage will cease, and cease completely.

[Dr. Murphy made a longitudinal incision along the inner side of the arm with the aneurysm as its center. Dissecting back the skin and superficial fascia and retracting them, left the aneurysm exposed in the center of the field, a blue-black, pulsating tumor, about like a Malaga grape in size, shape, and color. Lying directly against the aneurysm, at its upper border, was the median nerve.—Ed.]

The aneurysm shows up very nicely in the field. I have it fully exposed. There is the median nerve just above it, following the wall of the artery and of the aneurysm. If the patient were not a doctor and had not wisely come for operation as soon as he had convinced himself of the nature of this little tumor, he would before long have been compelled to seek operative relief for the neuralgia, which pressure on the median nerve would have caused as the aneurysm became larger. It is going to be difficult to separate the artery from the nerve as it is.

Visiting Doctor: Has the patient ever had any symptoms of pressure on the nerves?

Dr. Murphy: None at all. He has never had any numbness, tingling, or shooting pains in the hand or arm or any disturbance of motion.

[Dr. Murphy freed the aneurysm from the median nerve by a careful dissection and then dissected the former free from the other nerves and from the vein.]

It is a sacculated aneurysm. It looks to me at present as though it will be a suitable case for endoaneurysmorrhaphy. We shall be very happy if we can avoid ligation.

[Dr. Murphy applied the clamps to the artery on each side of the aneurysm. He opened the sac and found enough intact arterial wall to justify performing an aneurysmorrhaphy. Two plaques of atheroma were found on the thinned wall of the sac and appear to have been the cause of the weakening of the vessel. By inserting four mattress sutures, as shown in the accompany-

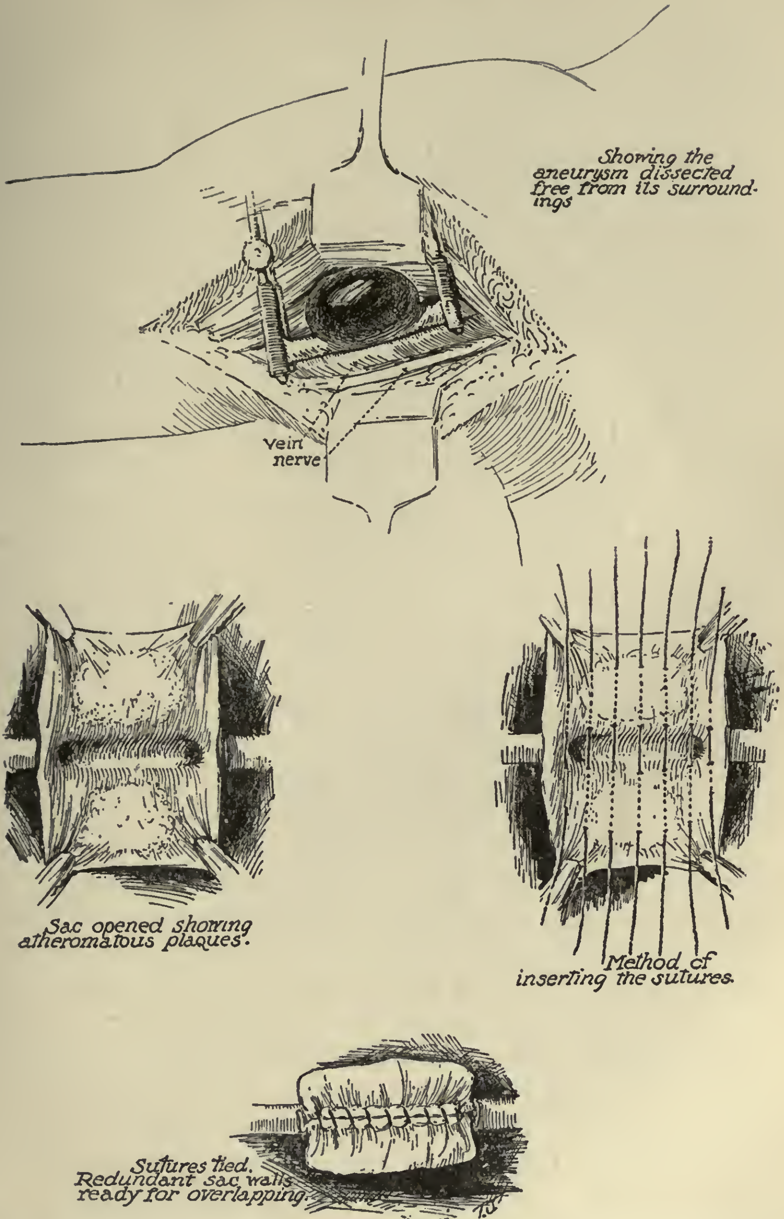

Showing the aneurysm dissected free from its surroundings
Vein
nerve
Sac opened showing atheromatous plaques.
Method of inserting the sutures.
Sutures tied. Redundant sac walls ready for overlapping.

Fig. 12.—Aneurysm of the brachial artery radically cured by endoaneurysmorrhaphy.

ing illustrations, a new arterial lumen was created and the excess of the sac-wall sutured over it. The suturing was so accurately carried out that there was not a drop of oozing after the clamps were removed, although the vessel pulsated normally in both its old and new portions.—Ed.]

Let the record show that the defect in the arterial wall was about ½ inch in length, and that the only vessel which it was found necessary to tie was a small anastomosing vessel opening into the sac.

Senn used to close these aneurysms by tying the sutures on the inside of the vessel, and these loose ends favored thrombosis, which is the great bugbear of all vascular surgery. I much prefer Matas' method. This sac-wall is as thin as a piece of paper. This is not a true aneurysm, I believe, because it seems to have only the adventitia left—none of the media. I should like to produce an endarteritis obliterans between the overlapping walls of the sac where I have placed them in contact. Such an obliteration of the space between the overlapping sac-walls, which are as thin as paper, would materially strengthen the external wall of the newly formed section of the brachial artery. I thought that the blood would probably shoot up in the air from the leaking stitches in the arterial wall when I removed the clamps and took off the compress; but I was very agreeably surprised to find not a single drop escaping, so that you have missed the lesson of seeing how quickly such a leak usually stops when judiciously compressed. This new vessel must stand a blood-pressure of 142, and so we are particularly gratified at the way our sutures are holding. If an endarteritis obliterans develops in the new lumen which we have just made, as well as between the layers of the superimposed sac, we shall not feel sorry, particularly if it will develop slowly enough to aid in the formation of a collateral circulation for the vessel, which should take about seventy-two hours to form. The collateral circulation develops very rapidly in every vessel in the anatomy after its blocking. We hope that the new lumen which we have made will stay open long enough to permit the development of enough collateral circulation to take care of the nutrition of the forearm and hand.

Let the record show that we performed an endoaneurysmorrhaphy: namely, that we sutured the entire coat of the artery, and that the opening in the artery was from ½ to ⅝ inch in length. We used a conjunctiva needle to carry the mattress sutures of fine silk, and we included the entire coat of the artery in the stitch; after having reëstablished the vessel lumen, we obliterated the exuberant wall of the aneurysm by suture. Both the brachial vein and the median nerve had to be separated from the wall of the aneurysm, the former being exposed for a distance of 3½ inches. The freeing of the nerve was more difficult and delicate a task than the freeing of the vein.

Those little spring forceps which we used here to shut off the flow in the brachial artery on each side of the aneurysm are those used commonly in operations on the muscles of the eye, and also for fixation of the sclera during operative procedures.

Let the record show that the dilatation of the vessel-wall occurred apparently not where the atheromatous plaques were located, but between and around them. We did not traumatize the internal coat of the artery in any part of the work which we have just carried out. These small vessel clamps which we used merely compressed the vessel lumen without traumatizing the intima. I found in my experimental vascular work that wherever I traumatized the artery in any way with the clamp, I always had trouble subsequently, chiefly endarteritis and thrombosis, but occasionally a consequent periarteritis made trouble, with finally a contraction and diminution of the vessel lumen.

In the Museum of the College of Surgeons in London there are a number of specimens showing a complete occlusion of the lower portion of the aorta as a sequence of Pott's disease, where the inflammatory process involved the outer sheath of the artery and finally resulted in a cicatricial contraction which closed the vessel altogether. In all these cases there was developed a sufficient collateral circulation because the gradual nature of the occlusion allowed plenty of time. The late Dr. Senn showed that if one could gradually diminish the flow through a vascular trunk, there would develop a thoroughly adequate collateral circulation. If this patient develops an endarteritis or a thrombosis in a week or ten days, and the vessel lumen slowly closes, that will be the best thing that could possibly happen to him. Why? Because the development of collaterals will take care of the circulation in the forearm, and a recurrence of the aneurysm is rendered impossible once the sac is obliterated.

This reconstructive operation which we have performed has a certain percentage of recurrences; but we have diminished the lumen of the artery, strengthened its weak area by lapping the layers of the sac over it, and have thus prepared the way for a collateral circulation to form. The collateral circulation is the key to success.

The paper, based on the experimental surgery which I did in 1896, was read at the International Surgical Congress at Moscow in 1897. I demonstrated there the first successful case of arterial suture, and showed that the principle governing the work was that the temporary reconstruction of the circulation thus secured lasted long enough for adequate collaterals to develop. We have had quite a number of cases of vascular suture since then, but have not reported them. My former associate, Dr. J. M. Neff, once collected them, and had them nearly ready for publication, but, other matters coming up, he never quite finished the article.

Carrel, when he was here in Chicago, took up with Guthrie the work of vascular suture where we left off. Carrel and Guthrie introduced the end-to-end suture of the vessels, making an ectropion of the intima. Our invagination method, while technically not so ideal probably as the end-to-end ectropion suture, possesses the great practical advantage of being so easy to carry out that any one who has had a little experience with arterial suture can perform it successfully. Use fine needles and double needle each thread. Draw the proximal vessel end to the distal end, invaginating it, and then top-sew the junction. In practice it gives a good result.

I operated my first case on the human being the day the corner-stone of the Alexian Brothers' Hospital was laid. I went from the ceremony there directly over to the County Hospital to perform the operation. The patient had a traumatic rupture of the femoral artery, and the distal end of the vessel was so turned to one side and pulled down by the profunda close to it that I had to make quite an incision in order to find it. After

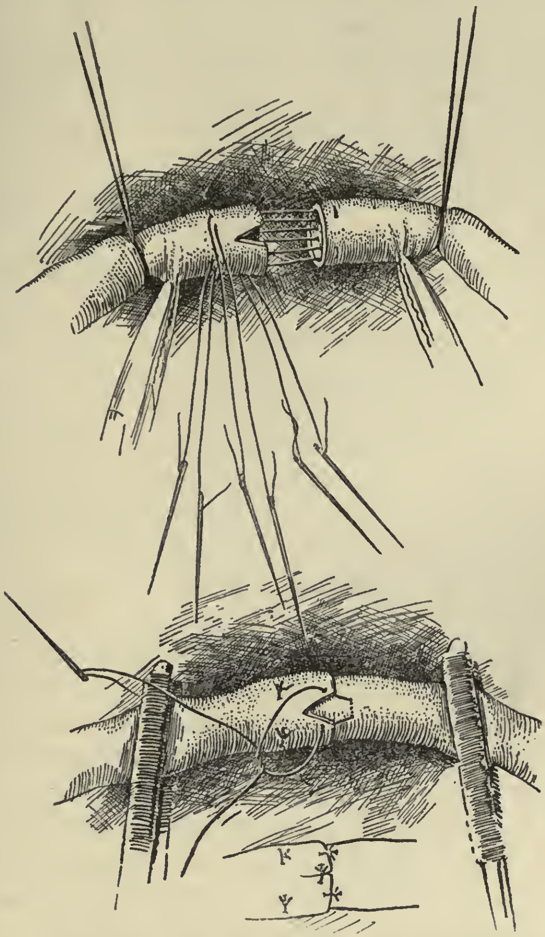


Fig. 13.—Dr. Murphy's method of performing vascular anastomosis by end-to-end invagination, as mentioned in the text. The upper drawing shows one method of controlling the hemorrhage and steadying the vessel-ends by ligatures gently tied; and also the method of inserting the three double-needled mattress invaginating sutures. The lower drawing represents the vessels compressed by rubber-covered vessel clamps, the invaginating sutures tied, and the approximation mattress suture being inserted. The outline drawing placed below shows all sutures tied.

the invagination the patient had an immediate restoration of pulsation in the posterior tibial at the ankle, and went on to an uneventful and perfect recovery. That was the first successful arterial anastomosis ever performed.

With a fine suture and a fine needle Carrel has succeeded in making a number of organ transplants by reuniting the proper vessels. Of course, such transplants will not functionate, because a transplanted organ, in order to live and functionate, must be not only from the same species, but also from the same individual. Therefore, while biologic difficulties, at present apparently insuperable, still stand in the way of successful organ transplantations, yet the technical difficulties of the transplantation have already been overcome.

Another procedure which has been carried out successfully where a considerable portion of an artery has been destroyed is to excise completely the damaged portion and insert in its place a section of a vein and suture the latter *in situ*. This type of substitution works very well if the suture is accurate. Often the substituted section of vein will heal permanently in place; but the important thing which it does is to reëstablish temporarily the arterial communication, and keep it open until a collateral circulation has time to form.

Endoaneurysmorrhaphy has come to stay. Dr. Matas, who has devoted much time to this line of work, has succeeded in establishing a thoroughly practical and well-recognized operation. It is astonishing that so valuable an operation has received so little recognition abroad. In the statistics published in 1909, there were recorded only three operations performed by foreign surgeons. All the others were performed by 45 different surgeons in America. No similar operation originated abroad could go so long unrecognized in our country. We Americans are little inclined to be Chauvinists. We are pretty apt to take what is good where we find it, and if we owe a great debt to foreign medicine, it is because we have had the breadth of vision and unbiased minds necessary to profit from it.

[Note.—Friday, September 18, 1914: The patient is up and walking about. He has a good radial pulse in the operated arm, and has had ever since the operation. He has no pain, numbness, pallor, or cyanosis in the forearm or hand, and no suggestion of any return of the aneurysm.

January 2, 1915: The radial pulsation has not been discontinued at any time since the operation. The patient has made an uneventful recovery.—Ed.]

DIVISION OF THE BRACHIAL PLEXUS AT THE LEVEL OF THE FIRST RIB.—SUTURING OF THE DIVIDED NERVE-TRUNKS

HISTORY

The patient, a boy aged fourteen years, entered the hospital October 15, 1914, with the following history: On June 29, 1914, while crossing a railroad track in a wagon, he was hit by a train and thrown about 40 feet. He struck the ground on his right side. He was unconscious for one week after the accident. After regaining consciousness he remembered nothing of the accident. He suffered from sharp pains in the hand from the wrist down into the fingers, and was unable to use the arm at all. He also had practically no sensation in the arm below the elbow, but sensation was intact from the shoulder to the elbow, or about one inch below. The arm was not swollen or red. He had a black and blue mark over the right shoulder and over the middle of the right clavicle and the neck above the clavicle. The right side of his head also was black and blue. His uncle, a doctor, noticed that the patient's right palpebral fissure was smaller than the left after the accident, and also that the right pupil was the smaller of the two. His vision was and is normal. He had no bleeding from the nose, mouth, or ears, and has had no convulsions at any time.

There is a constant feeling of numbness in the right hand at the present time when the arm is still. There is also a tingling sensation in the flexure of the elbow and on the inner side of the arm and hand. There is no power of voluntary motion in the arm.

COMMENTS AND OPERATION

Dr. Murphy (October 22, 1914): What is the matter with him?

INTERN: He has a complete paralysis of the arm.

DR. MURPHY: Due to what?

INTERN: To severing of all the motor nerves.

DR. MURPHY: Where?

INTERN: In the brachial plexus.

DR. MURPHY: If *all* the motor fibers of the plexus are cut, how does he move the muscles supplied by his circumflex nerve? How does he move his latissimus dorsi? How does he move his deltoid?

INTERN: He has some function of the sensory fibers.

DR. MURPHY: We are talking about motion, not sensation.

INTERN: He gets some response from the musculospiral group and from the circumflex group coming from ——

DR. MURPHY: Yes, where do they come from? What muscle does the musculospiral nerve supply that he can move? His triceps is out of commission, and all his forearm muscles. But he can move his biceps a little. Which branch of the musculospiral goes to the biceps?

INTERN: I did not say the musculospiral supplies the biceps.

DR. MURPHY: You implied as much. The fact is that you have not made any analysis of the muscles which are paralyzed or not paralyzed. How does the patient elevate his shoulder if all his brachial plexus is out of commission?

INTERN: I do not know.

DR. MURPHY: What muscle does the spinal accessory supply?

INTERN: The trapezius.

DR. MURPHY: What does the trapezius do?

INTERN: Lifts the shoulder.

DR. MURPHY: From what portion of the brachial plexus does that come?

INTERN: The spinal accessory ——

DR. MURPHY: Yes. Does it come from the outer cord?

INTERN: I do not know.

DR. MURPHY: You do not even know from which plexus it comes, apparently. You evidently did not go over the analysis of this case very carefully, did you?

INTERN: I went over it with Dr. Mix.

Dr. Murphy: But you evidently did not learn anything while doing so. You did not go back to your room, take down your anatomy, and go over the anatomic details to satisfy yourself as to the nature of the lesion present. What about the function of his phrenic nerve on the right side? What about the movement of his diaphragm? [No answer.] You see, doctor, how little you really know about this case.

The diaphragm does move; but the phrenic nerve comes from the third, fourth, and fifth cervical roots, and would be involved only if there were an evulsion of the third, fourth, and fifth cervical roots or a division of the phrenic nerve lower down. We will ask Dr. Mix to go over the neurologic aspect of this case.

Dr. Mix: The diagram on the blackboard shows you the brachial plexus, but, like many diagrams and most charts, it looks rather complex. It is necessary to have a diagram of the brachial plexus in your head if you are going to have a working basis for the unraveling of cases of this character. An easily remembered scheme of the formation of the brachial plexus is the following: The brachial plexus is made up of the anterior divisions of the fifth, sixth, seventh, and eighth cervical nerves and of the first dorsal nerve. These unite in this manner: the fifth and sixth unite to make the upper trunk; the seventh makes a middle trunk by itself; the eighth cervical and first dorsal nerves unite to make a lower trunk. Now each one of these three trunks divides into an anterior and a posterior division. We, therefore, have three anterior and three posterior divisions. The anterior divisions of the upper and middle trunks unite to make the outer cord of the brachial plexus. The posterior divisions of all three trunks unite to make the posterior cord of the brachial plexus. The anterior division of the lower trunk makes by itself the inner cord of the brachial plexus. We have, therefore, three cords—the outer, posterior, and inner. From the outer cord is given off the external anterior thoracic nerve, the musculocutaneous nerve, and the outer head of the median nerve. From the posterior cord there are given off the musculospiral and circumflex nerves. From the inner cord are given off the internal cutaneous, the lesser internal cutaneous, the ulnar, the internal anterior thoracic, and the inner head of the median nerve.

From the trunks of the plexus previous to their separation into three anterior and three posterior divisions, nerves are given off which supply the rhomboidei, the scaleni, the longus colli, and the levator anguli scapulæ. From the upper trunk is given off the suprascapular nerve, which supplies the supraspinatus and infraspinatus muscles. From one of the posterior divisions previous to its union with the other two to make the musculospiral and the circumflex nerves there are given off the long, middle, and short subscapular nerves. Another extremely important nerve of the brachial plexus going to the serratus magnus muscle, known as the posterior thoracic or the external respiratory nerve of Bell, comes from the trunks of the nerves previous to their division into anterior and posterior divisions. With this scheme of the brachial plexus in mind it is a rather simple undertaking to analyze the present case.

The next point to determine is, what neurologic defects does this patient show? At first glance his symptomatology may seem rather strange. His whole right arm is apparently paralyzed. Let us investigate first the condition of the individual nerves arising from the outer cord. These consist of the external anterior thoracic, the musculocutaneous, and the outer head of the median. What do we find? We find that the pectoralis major, which is supplied by the external anterior thoracic, is paralyzed. So also is the pectoralis minor in part supplied by the same nerve. We find, furthermore, that the biceps, the coracobrachialis, and the brachialis anticus, all of which are supplied by the musculocutaneous nerve, are paralyzed and atrophic. We will disregard the territory supplied by the outer head of the median for a moment.

Let us next examine the nerves from the inner cord of the brachial plexus. The motor nerves are the ulnar, the outer head of the median, and the internal anterior thoracic. The internal anterior thoracic aids in the supply of the pectoral muscles, which, as we have already seen, are completely paralyzed. The ulnar nerve supplies, besides cutaneous branches, the flexor carpi ulnaris, the ulnar half of the flexor profundus digitorum, all the muscles of the little finger group, the interossei and two outer lumbricales, the two adductors of the thumb, and half of the flexor brevis pollicis. All these muscles are paralyzed and atrophic. The median nerve, made up by the union of two heads, one from the inner and the other from the outer cord, supplies, as you will remember, all the flexor muscles on the front of the forearm (except the flexor carpi ulnaris), together with the pronator radii teres and the pronator quadratus, as well as the two remaining lumbricales and the two one-half muscles of the thumb not supplied by the ulnar, namely, the abductor pollicis, opponens pollicis, and one half of the flexor brevis pollicis. All these muscles are paralyzed and atrophic.

Turning to the posterior cord, we find that it gives off the circumflex and the musculospiral nerves. The circumflex, it will be remembered, supplies the deltoid and the teres minor. Both of these muscles are paralyzed and atrophic. The second branch of the posterior cord is the musculospiral. This supplies the triceps muscle, the supinators, and all the forearm extensors either by itself or by means of its posterior interosseous branch. These muscles are all paralyzed and atrophic. It is, therefore, evident that this patient *has no activity in a single muscle* supplied by any of the nerves coming from the inner, posterior, or outer cords. In other words, he has absolutely no power in his triceps or in the muscles on the front of the arm—not a single trace of response in any of the muscles of the forearm or hand. Furthermore, when you examine the hand, you will note that he has the claw-hand so characteristic of a paralysis of the ulnar nerve.

Now as to his sensory supply. This is also interrupted in a peculiar way. He has no sensation distal to the region of the elbow except for a distance of about 2½ inches behind and one inch in front. Below this line he is absolutely unable to tell when he is touched. Not only is his sense of touch absent, but he has neither pain sense nor thermal sense in the skin below that line. This fact would indicate, therefore, that there is some sensation in the territory supplied by his internal cutaneous nerve, his lesser internal cutaneous, and in some of the territory of supply on the back of the arm of the musculospiral. Some sensation is present, but it is only protopathic sensation. One would think that if he had so great a loss of power in his muscles he ought to have a correspondingly large defect in the distribution of cutaneous sensation. Curiously enough it is not co-extensive with the motor loss.

Let us now turn to those branches of the brachial plexus which are often lost sight of, but which I mentioned a few moments ago. The muscles which are supplied by the brachial plexus before it forms the three cords and gives off this group of nerves are the latissimus dorsi, the subscapularis, the supraspinatus and the infraspinatus, the teres major, the rhomboidei, the scaleni, the longus colli, the levator anguli scapulæ, and the serratus magnus. Let us examine these muscles. We found on testing the boy that he was able to push remarkably well with his right shoulder. This means that his right serratus magnus was innervated powerfully. Indeed, his ability to push with his right shoulder was even better than his ability to push with his left. Now the serratus magnus or the pushing muscle is supplied by the posterior thoracic nerve, or, as it used to be called, the external respiratory nerve of Bell, since the serratus magnus is an accessory muscle of respiration. His latissimus dorsi, his subscapularis, and his teres major muscles were also found to be intact. Indeed, his latissimus dorsi was very strong. Therefore, it is rather evident that the nerve-supply to these muscles, which are not atrophic and are not paralyzed, is intact. These muscles are supplied by the long, middle, and short subscapular nerves, which are given off from the posterior divisions of the trunks previous to their union to form the posterior cord. This would indicate that the upper portion of the brachial plexus is still intact.

Examination also shows the levator anguli scapulæ, the scaleni, and the rhomboidei to be well innervated. These muscles, too, are supplied by nerves leaving the brachial plexus previous to the formation of the cords. Lastly, the supraspinatus and infraspinatus, supplied by the suprascapular nerve, are not paralyzed. Evidently, then, the brachial plexus must be interrupted in its course after it has given off the branches which supply all these muscles. The last great muscle which is intact is the diaphragm. So far as I was able to determine, the boy's diaphragm on the right side moves perfectly well. This means an intact phrenic nerve. The phrenic, as you know, receives an important communicating branch from the fifth cervical nerve previous to its joining the sixth to form the upper trunk.

Now that we have located the level and extent of the injury, the next question which presents itself is, what happened? Was his clavicle jammed back against the first rib by the violence of the accident, nipping the brachial plexus between the two bones, or was the brachial plexus torn out of the intervertebral foramina by its roots? Even without analyzing the anatomic possibilities closely, there is one point which may be settled at once. Whenever the brachial plexus is torn out by the roots the first dorsal nerve escapes. The fifth, sixth, and seventh nerves may be pulled out, but it is not easy for an accident to tear out by the roots the eighth cervical and the first dorsal nerves because of the protection afforded them by the clavicle and the first rib. The eighth cervical and first dorsal nerves make up the lower trunk and also the inner cord of the plexus. In evulsion injuries the nerves having their origin from this portion of the plexus usually remain intact. But they are not intact here: they are extensively involved.

In this boy's case the musculocutaneous, median, circumflex, musculospiral, and ulnar territory is completely paralyzed. Therefore, one would say that, in all probability, the injury was not due to an evulsion, in which case the supraspinatus, infraspinatus, subscapularis, latissimus dorsi, teres major, serratus magnus, scaleni, rhomboidei, longus colli, and levator anguli scapulæ would *not* have escaped, but rather to a cutting in two of the brachial plexus, in which case the musculocutaneous, circumflex, musculospiral, median, and ulnar nerves *would* be involved, as we find actually to be the case. This fact leads one to the belief that in this patient the diagnosis of evulsion is open to grave question.

Furthermore, this analysis leads one to hope that perhaps the difficulty was the pinching of the brachial plexus between the clavicle and the first rib, producing an injury to the cords which lie immediately behind the clavicle.

You will remember that the trunks of the brachial plexus lie between the scalenus medius and scalenus anticus muscles. The three cords of the brachial plexus lie behind the clavicle, and the nerve-trunks issuing from the three cords are given off just below the clavicle. In this boy's case all the nerves given off by the brachial plexus below the clavicle are interrupted; all three cords behind the clavicle are involved; but *above* the clavicle the long middle and short subscapulars, the suprascapular, the posterior thoracic, and the phrenic undoubtedly have escaped injury. This fact, therefore, leads one to the almost inevitable conclusion that the injury to the plexus must have occurred behind the clavicle and below the point of emergence of the trunks from between the scalenus medius and scalenus anticus muscles, and that the lesion is not an evulsion, but a crushing of the trunks. An unusual feature of the case, however, is that the boy's clavicle was not broken when the violence was sufficiently great to have produced this lesion, although a fracture is not essential to produce pinching between the clavicle and the first rib. Moreover, since the sensory paralysis is not so absolute as the motor, possibly an absolutely complete division of the three cords did not take place.

It is not possible in this case to state exactly what has to be done surgically. Everything points to the more or less complete division of the great cords of the brachial plexus; but, on the other hand, the boy's clavicle has not been broken. You will remember from the intern's history that the side of the boy's head, his shoulder, and neck were badly bruised. This statement harmonizes very well with the diagnosis of subclavicular division of the plexus notwithstanding the absence of signs of fracture of the clavicle; moreover, we cannot dodge the fact that this boy can use his subscapularis, his supraspinatus and infraspinatus, his serratus magnus, and his latissimus dorsi muscles, and that he also has the normal power in and the use of his diaphragm, his levator anguli scapulæ, his rhomboidei, and scaleni.

When one bears in mind these facts, and when one also recalls the fact that these muscles are supplied by the branches given off by the cervical roots before they go to form the trunks of the cord, then one becomes fairly certain that there is hope for this boy in an operation. There would be practically no hope whatever of recovery if a complete evulsion of the roots of the brachial plexus had occurred.

In this case there is still one more difficulty in arriving at an absolute conclusion. The patient has a slight enophthalmos, the right bulb being distinctly retracted. He has also a narrow right palpebral fissure and a right pupil which is smaller than the left. These three symptoms, enophthalmos, narrow palpebral fissure, and small pupil, are signs of an involvement of the right cord of the sympathetic nervous system in the neck. In other words, these symptoms give us the impression that the right cervical sympathetic cord has been hurt by this accident. Whether it was hurt independently of the injury to the brachial plexus or whether it was affected by reason of its connection by filaments with the brachial plexus, I cannot say. This is a matter that will have to be worked out at the time of operation, if possible. His uncle, who is a physician, was the first to notice the inequality of the pupils. Our intern noted the narrow palpebral fissure and I found both of these symptoms, together with the enophthalmos, when I examined the patient; so that by three medical observers coöperating we managed to bring together the three signs which indicate a disturbance of the cervical sympathetic cord. This involvement of the sympathetic gives one the conviction that the disturbance in his neck must have been a deep-seated one.

Our conclusion is: That there was no evulsion of the nerves of the brachial plexus, but division behind the clavicle of the cords of the plexus, and an escape from injury of the nerves which supply the serratus magnus, the latissimus dorsi, and the scapular group; furthermore, that, on account of the pupillary disturbance, the enophthalmos, and the narrow palpebral fissure, there was some injury done his right cervical sympathetic cord.

Dr. Murphy: One of the important factors in the mechanism of an injury such as this one is the anatomic construction of the openings in the vertebral column through which these nerves pass. Each of the cervical nerves passes over the anterior surface of the transverse process of a cervical vertebra. This bony surface ends abruptly—often in a distinct ridge. Keeping this anatomic point in mind, it is easy to see how a sharp-edged foreign body passing down the neck over these ridges with considerable violence could shear off all the nerves which go to make up the cervical and brachial plexuses.

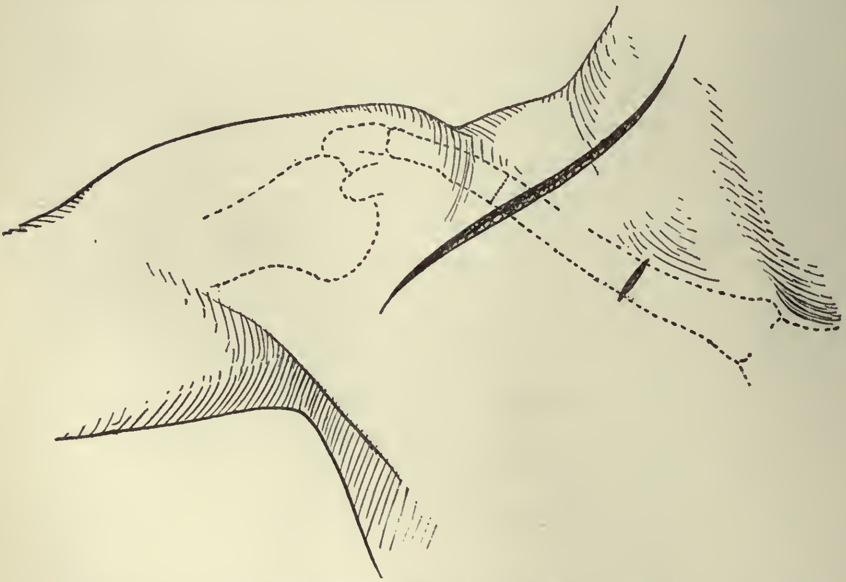


Fig. 14.—Division of the brachial plexus at the level of the first rib. Suturing of the divided nerve-trunks. Note the line of incision, the upper half of which was made first for the exposure of the foramina of exit of the nerves from the vertebral column. This incision was then extended downward over the clavicle nearly to the lower border of the pectoralis major. Through the incision the clavicle was divided, as shown by the dotted line, and then cut a second time by a costotome through the short incision internal to the main cut. The clavicle was then turned inward and the plexus exposed.

We are exposing the scalenus anticus muscle by an incision made parallel to its outer edge, and are using it as a guide to the location of the plexus, which, we know from our anatomy,

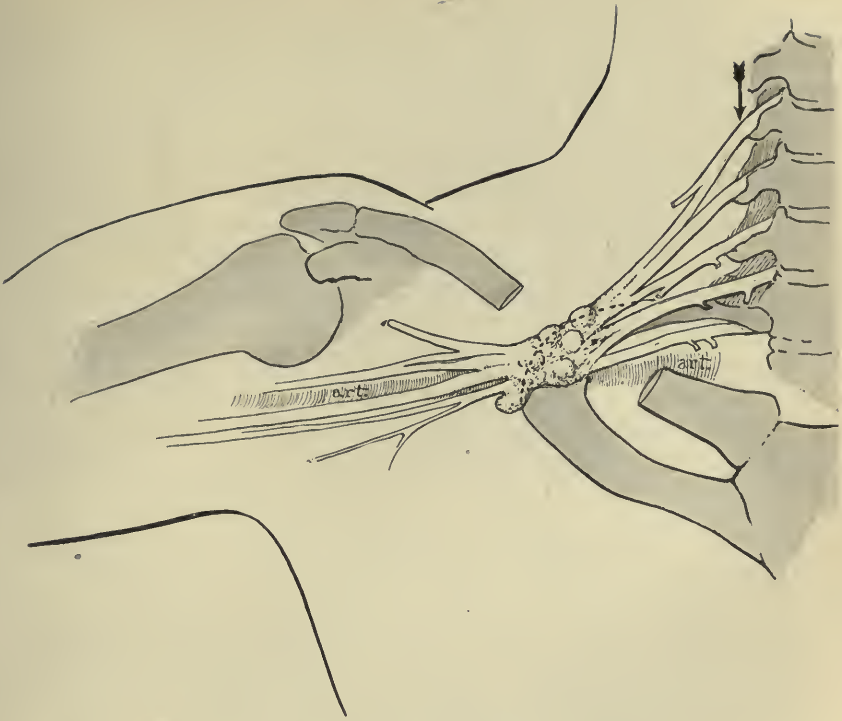


Fig. 15.—Division of the brachial plexus at the level of the first rib. Suturing of the divided nerve-trunks. This diagrammatic drawing shows the pathologic changes found at the site of the division of the plexus over the first rib. It also shows how the nerves going to make up the plexus pass over the sharp edges of the transverse processes of the cervical vertebræ after leaving the spinal canal. Note how easily either a blunt or a sharp weapon striking the side of the vertebral column forcibly in the downward direction indicated by the arrow could shear off the cervical nerves before they join to form the cervical and brachial plexuses. Of course, the upper two or three cervical nerves are considerably protected against such an injury by the projecting angle of the jaw and the last nerve by the presence of the clavicle and first rib. Note the terminal neuromas embedded in a mass of scar tissue which also embraced the subclavian vessels and made the subclavian artery difficult to dissect away from the cords of the plexus. The upper neuromas appeared distinctly larger than the lower when they were finally dissected free from the enveloping cicatricial tissue.

lies directly behind it, between it and the scalenus medius. An important question which only our operative exploration can decide is whether the nerves are cut, compressed, or evulsed from the foramina of exit.

[Dr. Murphy exposed the fifth, sixth, seventh, and eighth cervical nerves at their points of emergence from the intervertebral foramina. The nerves were found in place and showed no signs of any neuroma formation. Thinking that they might have been torn inside the vertebral openings, an attempt was made to withdraw them by gentle traction, but in vain, thus showing them to be intact at this level. The faradic excitor was applied to the nerve-trunks, but without eliciting any muscular response in the arm or hand. The conclusion was, therefore, unavoidable that the lesion lay further distal in the plexus, probably behind the clavicle.

Accordingly, the skin incision was continued downward over the clavicle toward the axilla, nearly to the lower border of the pectoralis major; the clavicle was cut at two places, the outer with the Gigli saw and the inner with the costotome, after making a preliminary short nick with a scalpel through the skin over the clavicle (see the accompanying illustration), and the cut ends were turned out of the field, exposing an area reaching from the vertebral column into the axilla. The subclavius muscle was cautiously divided over the plexus, and likewise the pectoralis major and minor tendons. The plexus and vessels then lay exposed, and the latter were carefully avoided as the cords of the plexus were isolated and found to run into a mass of cicatricial tissue. On account of their involvement in the latter it was very difficult to identify the individual cords and nerves. After first carefully isolating the subclavian artery and vein, the trunks and nerves were dissected out of the mass of scar tissue. It became apparent that all the trunks of the brachial plexus had been divided between the clavicle and the first rib, the former probably having been forced down violently on the latter at the time of the original injury. Consequently, the different trunks were cut at different levels. The only fibers of the plexus which remained intact were those running to the serratus magnus and the latissimus dorsi. When the ends of the trunks were exposed above and below, neuromas were found on them. The latter were excised far enough back on the nerve-trunks so that the individual fasciculi stood out on the cut surfaces like brush-ends. The nerve-ends were then accurately approximated with fine catgut sutures, no very great attempt being made to identify the individual trunks as they were reunited, since we know that the return of function will be excellent, even though the trunks are not connected up exactly as they stood originally. The distal end of one of the nerve-trunks—apparently the inner cord—was not only turned to one side, but also actually bent back on itself, so that the terminal neuroma pointed distalward instead of proximally, and confused the anatomic picture until Dr. Murphy had it dissected free from its bed of scar tissue.—Ed.]

Dr. Murphy: This may seem like a lot of work, and tedious work at that, which we have been doing to free and reunite this plexus; but if this were *your* boy, it would not seem too much. We are patient because we are working for an arm and we hope to secure a successful result. We have had two other plexus injury cases, almost exactly like this one, and we secured perfect results for them. Therefore, we are all the more hopeful for this youth.

One of the two previous cases was a porter whose business was and is to handle trunks. He was shot from above and behind. The bullet passed under his clavicle and mowed down his brachial plexus. We sutured the freshened ends of the cut nerves just as we have been doing here, and the man is now back handling trunks as easily and at as good a salary as ever.

The other patient was an Austrian laborer. We operated him on the twenty-third day of December, 1910, and secured a perfect result. Two years later he walked from his home town in southern Indiana and reached here the twenty-fourth of December, to celebrate the anniversary by kissing my hand for having given him a perfectly useful arm. He could not speak one word of English, but he didn't need words to express his gratitude. That is what can be done for a complete paralysis due to a plexus lesion. [See previous issues of the Clinics.]

Visiting Doctor: How would you bridge the gap if the two ends of the divided trunk could not be made to meet?

Dr. Murphy: I should join them to the nerve either next below or next above, whichever happened to be the more convenient.

There was so much dissecting and handling of tissues done here that I rather like to apply a sponge wet with the old-fashioned 5 per cent. carbolic acid in order to stimulate the processes of repair and destroy any microörganisms which we may have introduced on our gloves or instruments.

We are suturing the clavicle back in position, and also this scalenus anticus muscle, as well as reuniting the subclavius muscle and the pectoralis major and minor tendons. We are leaving in a gutta-percha drain which runs up along the pectoral muscles toward the clavicle. We are taking all the tension off the skin by using a subcutaneous continuous catgut suture, so that there will be no traction on the horsehair skin sutures subsequently.

The tension on the sutured nerves was not great in this case, but we have ordered the arm dressed across the chest, in order to relieve whatever slight tension may be present.

Now, for the matter of regeneration! All cut peripheral nerves except the nerves of special sense will unite and function, if joined end-to-end after cutting off any mass of connective tissue which may be interposed. This result is obtained just as regularly as union is obtained in joining the two ends of a broken bone. If one merely sews the nerve-ends together after they have been divided for a number of weeks, without excising the connective tissue contained in and covering the terminal neuromas, one will secure no genuine nerve union at all and no reëstablishment of function will ensue, because the nerve tissue, the neurons, in such a case have no opportunity to become continuous. It was this fact which divided the profession into two camps for so many years on the subject of nerve anastomosis, one camp claiming that a nerve could be made to unite and function after being divided, and the other that it could not. The two schools were not equally divided on the subject, because 80 or 90 per cent. of doctors then believed that in the late cases, at least, there was no reëstablishment of function whatever possible. The reason why many of the older operators did not secure a reëstablishment of function in the late cases was because the nerve-ends at these late dates are capped with connective tissue, and these old surgeons did not take the pains to cut this scar tissue away and expose the nerve fasciculi. The peripheral nerves unite both early and late. They may unite as late as twenty-six years after they have been divided, because we have ourselves had a successful result after such a length of time had elapsed.

VISITING DOCTOR: How soon will there be evidence of regeneration?

DR. MURPHY: The period differs in different cases, for some reason which we do not at present thoroughly understand. Sometimes a motor nerve may regenerate as early as six weeks after suture, and the sensory nerves likewise; but the rule is that three or four months usually pass before the patient commences to get much motion. For a long time it was believed that nerve paralysis which had existed for a long time resulted in complete atrophy of the muscle-cells amounting to practical destruction. The muscle-cells *do* atrophy somewhat, but they are not destroyed. As soon as one reunites the nerve the muscle-cells commence to function again, and develop strength relatively rapidly. There seems to be no time limit in this direction either.

VISITING DOCTOR: What is the after-treatment?

DR. MURPHY: Nothing. Let it alone.

VISITING DOCTOR: Any massage?

DR. MURPHY: Nothing at all for six weeks. Then let the patient commence to use the limb passively and actively, and apply massage and electricity, if you will.

[NOTE.—The wound healed by complete primary union and the clavicle reunited promptly. The patient left the hospital six weeks after the operation and has not yet returned for reexamination.—ED.]

MIXED ROUND- AND SPINDLE-CELL PERIOSTEAL SARCOMA OF THE RIGHT FEMUR. —DISARTICULATION AT THE HIP

HISTORY

THE patient, a man aged thirty years, entered the hospital October 7, 1914. He has had the usual diseases of childhood. He denies all venereal infection. His family history is negative as to tuberculosis and malignant tumors.

The patient dates his present trouble back to November, 1913. While walking across a room in the dark he bumped into a stove, striking the anterior portion of his right thigh very sharply against it. He suffered no visible bruise, and felt no soreness during the next few days. He was able to walk after the accident with no particular inconvenience.

He had no further trouble in the limb until December 1, 1913, about a month later, when he noticed shooting pains through the upper part of the right thigh. These pains came on intermittently at first, occurring about twice a week; then they gradually increased in frequency until they became almost continuous by February 1, 1914. In April, 1914, in addition to the pains, he noticed a hard swelling appearing on the inner side of the upper third of the right femur. This swelling, which proved to be a tumor, was removed May 18, 1914. The patient was in that hospital ten days at the time of this operation, and gradually regained his former strength within a few weeks after leaving the hospital.

On July 11, 1914, the pains in the upper thigh returned as before the operation. An *x*-ray picture taken at this time showed a recurrence of the tumor, the patient states. Since then the pains have increased in severity and the tumor has grown quite rapidly to its present size. The patient has lost 25 pounds in weight in the past ten months. Otherwise he feels in his usual good health.

COMMENTS AND OPERATION

Dr. Murphy (October 8, 1914): We had the specimen removed for diagnosis at the previous operation and sent to us by the patient's surgeon, examined by two pathologists, both of whom stated that it was a round-cell sarcoma originating from the periosteum. The *x*-ray plates confirm this diagnosis.

Notwithstanding the fact that the diagnosis was made positively in April, that the clinical history was typical, and that the specimen examined by the pathologist was pronounced a sarcoma, the patient has gone all this time without allowing any effort to be made at saving his life. He comes to us now to let us amputate as a last resort—a last and faint hope. Of course, it is unfortunate, and it is unfair of the patient, to himself and to us, to have allowed that period of time to elapse before acting on his surgeon's recommendation. When the specimen of tumor was removed and examined and the diagnosis of sarcoma verified, there was only one sensible thing to do, and *then* was the time to do it. That was to amputate the limb. Any further waiting was merely tempting Providence. When he came back a few days ago he asked me what was my prognosis. I replied that nobody could tell for certain at this time whether those malignant cells, disturbed by the operation in May, have gone through the blood to other positions in the body or not. At any rate, physical and *x*-ray examination shows that he has no foci of demonstrable size in the lungs, which are the most frequent seat of metastases from osteal sarcomata.

Sarcoma occurs throughout the entire range of life, and with approximately equal frequency in every decade. Perhaps it is found a little more often in the third decade, that is, from twenty to thirty years of age, than in any other.

In my experience sarcomata of bone have been the most frequent. Dead-house statistics would, I think, show intrathoracic sarcoma, that is, sarcoma of the lung and mediastinum, to be more common on the autopsy table than bone sarcomata;

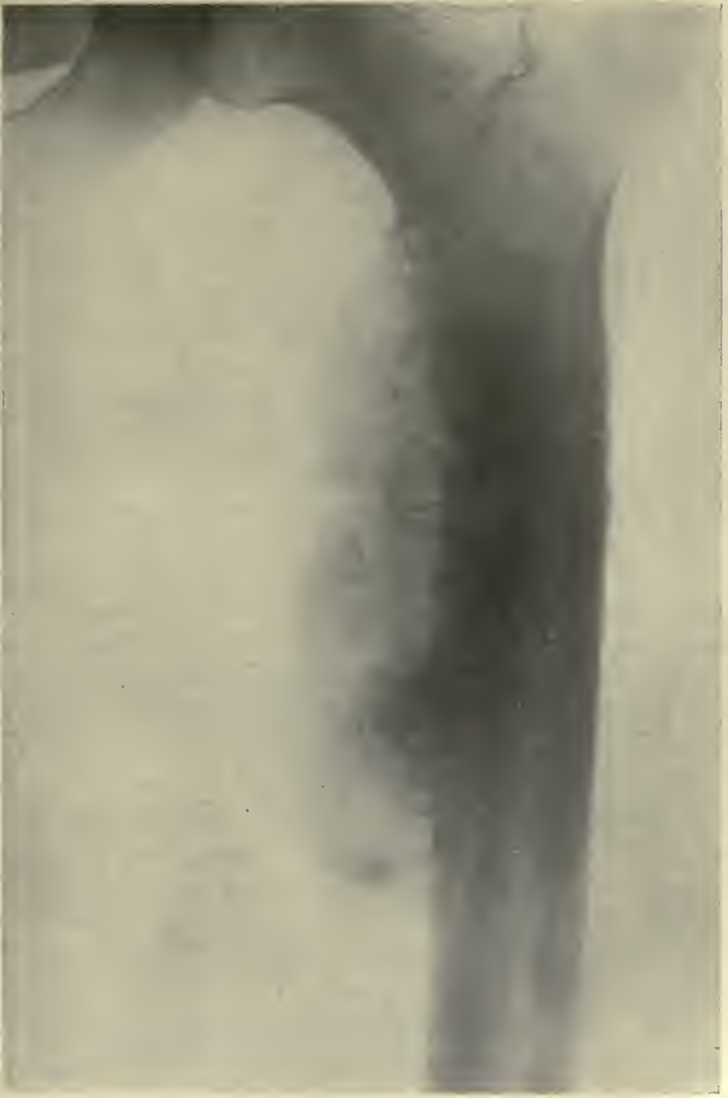


Fig. 16.—Mixed round- and spindle-cell periosteal sarcoma of the right femur having its origin on the inner side of the femur and extending into the adductor group of muscles. Anteroposterior roentgenogram made before operation. Note the radiating columns of bone sticking straight out from the femur through the tumor-like thick bristles. A roentgenologic feature characteristic of periosteal sarcoma.

but the former do not often seek their relief from the surgeon, since he has not much to offer them, whereas the latter are our frequent visitors.

Sarcomata are, beyond any doubt, the most frequent tumors of the bones. Although carcinomata and nephromata are more often found as secondary tumors in bone than are sarcomata, yet the great frequency of the latter as primary tumors gives them the predominance in any large collection of statistics. Some of these bone sarcomata are soft, particularly certain intramedullary growths; but the majority are firm or hard, and the hardest of these are the varieties which produce osteoid tissue and new bone. Such osteosarcomata do not necessarily have their primary seat in bone, for they are also found occasionally in the lung, the parotid gland, and elsewhere; but they are usually of bone origin.

Sarcoma, as a general proposition, tends to remain a local disease longer than does carcinoma. I have collected 31 cases of sarcoma observed at autopsy. Sixteen of them had no metastases demonstrable at the time of death; that is, over 50 per cent. of them were still a local disease, presumably eradicable by operation. How much greater must have been the percentage of curable cases at the time when the disease was first noticed!

Basing our practice on these facts, it has been our custom for some years now to perform only local removal of the tumor with the neighboring bone and soft tissues in the case of osteal sarcomata with no demonstrable metastases, wherever practicable and where the cell type was not of the most malignant character. Later we make a transplant, usually from a tibia, to fill the defect. In most cases this local operation accomplishes as much as an amputation, and, what is still more important, the patient consents to operation at a much earlier date than when amputation is advised. When one says to the patient, "I am going to take out the tumor with four, six, or eight inches of the femur, and put back just as long a piece from the tibia to take its place, so that the femur can grow on to full size to support the weight of the body," the patient will consent almost immediately when he will not consent at all to an amputation.

The cases which died of the disease after operation are much more likely to perish from distant metastases than from a local return of the disease. Not only is this fact true of bone sarcomata, but also of carcinoma metastases in bone, a few of which we have already excised when they were single, and the only demonstrable metastases.

Let me cite a case along this line which is fresh in my mind, because he died only a few days ago. His disease started as a sarcoma of the trochanteric zone of the femur, which developed after a slight injury of the femur. We saw him first some two or two and one-half years after the accident, when the growth was already moderately advanced. We felt able to assure him that a removal of the local disease by resection would be practically as safe against recurrence as an amputation, because the growth appeared still entirely encapsulated. We excised the upper end of the femur on the sixteenth of October, 1911, that is, three years ago. Up to the time of his death he had no return of the growth in his femur, but he developed metastases in his lungs, and they caused his death. In the 31 autopsy records of fatal sarcomata which we collected metastases in the lung occurred in only three out of the entire 31 cases. That is a very much lower percentage than we should expect from our experience, in which lung metastases have been very frequent. If we could have performed that excision of the upper end of the femur in the early days of his disease,—in the first months, for instance, as soon as the *x*-ray plate showed that he had a sarcoma,—how much better would have been his prospects of obtaining a permanent cure!

Of course, sarcomata differ very materially, not only in their gross microscopic appearances, but also in their conduct. Some sarcomata are practically benign. I can mention four types of giant-cell sarcomata of the mouth and nasopharynx that are very benign. One is the soft epulis, which begins in the gum on the side of a tooth. That is very benign. One can cut it out and cut it out and cut it out, and although it recurs it does not become malignant in the sense of forming metastases. These tumors often have their origin from a portion of an alveolar process or its periosteum and are, therefore, not cured until their seat

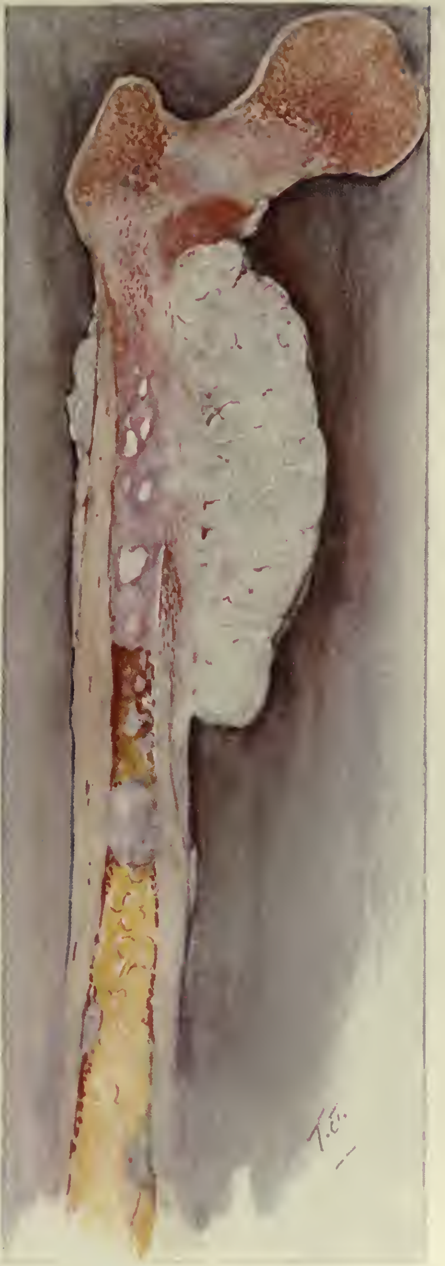


Fig. 17.—Round-cell periosteal sarcoma of the right femur. Specimen removed by exarticulation of the limb at the hip. The femur has been sawed open longitudinally. Note that while the spread of the growth has been principally outward into the soft parts, the medulla of the bone is already extensively involved, both by direct extension through the cortex of the bone at the level of the primary growth and also by lymphatic transmission through the marrow. The

of origin also is removed. Some surgeons remove the adjacent tooth, but one does not need to do more than remove the piece of bone from which the epulis grows.

Another benign but rare type is the giant-cell sarcoma occurring on the superior turbinate bone. It grows slowly, but recurs and recurs after operation. I recall a case I saw von Bergmann operate, which had been operated 118 times previously. von Bergmann went after it radically, split the nose, fractured the nasal bone, and exposed the periosteum from which the tumor originated, excised the tumor, and the periosteum with it, thus thoroughly extirpating the disease.

Another type is represented by a case I once saw, a giant-cell sarcoma near the opening of the sphenoid sinus. I saw it many years ago, and it remains in my mind as a giant-cell sarcoma. We did not know about chordoma developing in this locality in those days, or about hypophyseal remnants forming tumors around Rathke's pouch, because the field of hypophyseal surgery—which A. E. Halstead and Allen Kanavel opened up when they published their operations, some modification of which is now used by nearly all operators of the hypophysis—was then still a sealed book. At any rate, it was a tumor which recurred locally several times, until a radical removal with the underlying bone was performed.

The last and most common variety of these mild buccal sarcomata is the giant-cell epulis arising from the periosteum or the medulla of the jaws.

[As a preliminary step in the operation, Dr. Murphy ligated the external iliac vessels, both artery and vein, just at Poupart's ligament. Two ligatures were applied to each vessel, ½ inch apart, and the vessels divided between them. The artery had an additional ligature placed one-half inch above the first, for safety's sake, and an artery forceps was placed on the proximal side of the proximal ligature and was allowed to remain until the approximation of the amputation wound. The pressure from this artery forceps fractures the intima and allows the thrombokinase to escape from the endothelial cells, according to the current physiologic view at the time I originated the method, and this procedure favors the formation of a thrombus on the proximal side of the ligature. This insures us against subsequent hemorrhage, no matter what fate may befall the ligatures. We demonstrated the usefulness of this procedure in our experiments on vascular surgery in 1896, and we have used it in closing all large arteries since that time and have never had a secondary hemorrhage. The glands beneath Poupart's were found somewhat enlarged, and a few of them were removed for microscopic examination. Dr. Murphy made a longitudinal incision over the great trochanter and divided the capsule of the hip-joint widely, exposing the head and neck of the femur. The head was disarticulated from the acetabulum with comparatively little bleeding. The muscles connecting the femur with the pelvis were cut wide of their femoral insertions. The sciatic nerve was exposed, and, after being infiltrated with novocain, the proximal stump was split longitudinally for a distance of ¾ inch by an L-shaped division, and the axons reunited end to end, thus the better to insure against the formation of a painful neuroma. After completing the separation of the limb from the stump and ligating the bleeding points, the opposing muscle groups were united across the acetabulum so as to obliterate the dead space.—Ed.]

An amputation is always an admission of failure from a doctor's standpoint. An amputation is always death of a part, and signifies a physician's failure to save that part. It is never an achievement. We have not performed a typical amputation in this case; it has been more of a dissecting operation, because the muscles have been infected here more or less, either by the natural growth of the sarcoma or by the dissemination of sarcoma cells following the previous operation on the tumor. Therefore, we have attempted to take out all the adductor muscles up to their pelvic attachments.

In ligating the large vessels in such a wound I usually follow our custom of the preantiseptic era and tie them at a distance from the main wound, so that if the latter should become infected, there is no danger of a secondary hemorrhage taking place, as it might if the vessels had been ligated in the wound. Most of you men present to-day did not live in the secondary hem-

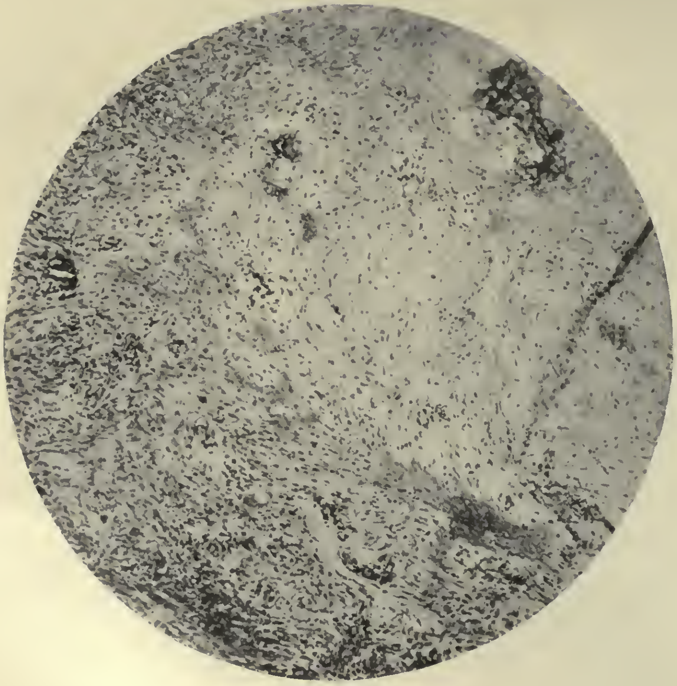


Fig. 18.—Mixed round- and spindle-cell periosteal sarcoma of the femur. This area shows the predominating cell type and, at the edge of the field, a small area of cartilaginous metaplasia with some areas of calcification (magnification, 60 diameters).

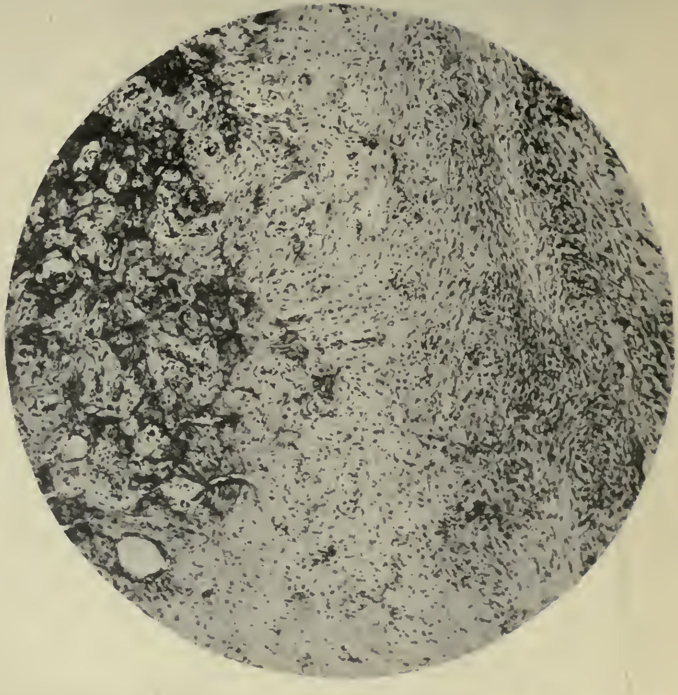


Fig. 19.—Mixed round- and spindle-cell periosteal sarcoma of the femur. This area shows chiefly spindle-cell strands with a focus of calcification at one side (magnification, 60 diameters).

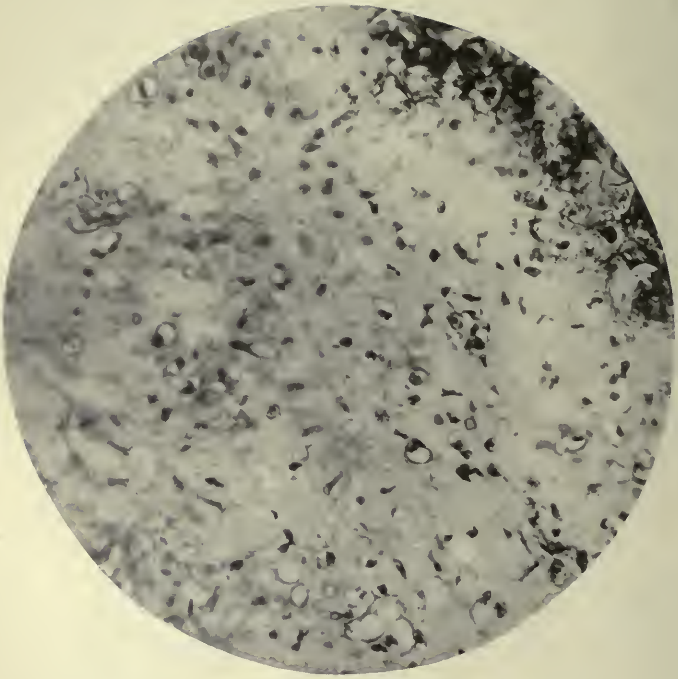


Fig. 20.—Mixed round- and spindle-cell periosteal sarcoma of the femur. This area shows cartilaginous metaplasia with calcium deposits at the extreme edge (magnification, 200 diameters).

orrhage period, preceding the antiseptic and aseptic eras, as I did. If you had, you would have a very wholesome respect for secondary hemorrhage still with you. Although I tied the iliac vessels outside the main wound, I tied the obturator inside it—as I should certainly not have done in my preantiseptic practice. It was not a large vessel here.

We sewed the split halves of the sciatic nerve together so as to secure an approximation of the neurons. By joining the two halves in that way the least possible amount of extraneous tissue comes to lie on the end of the nerve. The axons are covered so they cannot become surrounded by connective tissue. We do everything possible to lessen the likelihood of outside scar tissue forming. We cap those ends with the soft tissues, so they will not become involved in a cicatricial mass, so that the patient will have little or no tendency to the formation of a neuroma, and less likelihood subsequently of feeling pain referred to his foot or his femur. Pain after amputation is often referred to areas around a joint, and this pain subsequently may become one of the most annoying complaints that can develop after an amputation.

The pathologist reports that the glands removed are infected with a round-cell sarcoma, a statement which we do not accept without a considerable mental reservation. You know how difficult it is often to tell an acutely infected gland from a round-cell sarcoma in a gland. It is a question in my mind whether these glands are not enlarged possibly from bacterial irritation following the previous operation. We shall have the glands further studied with paraffin sections, and shall also have a careful study made of different areas from the original tumor in the femur. If the cell types correspond, we shall accept the pathologist's report; otherwise we shall not. From my gross examination of the glands they looked more like a simple hyperplasia, the result of a mild infection, since the glands were nowhere adherent to one another or to the surrounding tissues, and the cut surface of the glands showed the gray color of a lymphatic hyperplasia rather than the motley appearance of a sarcoma metastasis.

We are closing the skin with horsehair. The rubber drain which we have inserted extends through the entire field. [The usual copious nosophen gauze dressing was then applied.]

[Pathologist's Final Report (Dr. W. O. Sweek).—The growth in the femur is a mixed round- and spindle-cell periosteal sarcoma, with numerous areas of cartilaginous and osteoid metaplasia. A more careful study of a series of sections of the lymph-glands excised and a comparison of them with the cell types of the original tumor lend strength to Dr. Murphy's view, based on the gross appearance of the glands that they represent a simple inflammatory hyperplasia rather than an invasion by the sarcoma.]

[Note.—Complete primary union. The patient has had no neuralgic pains following the operation. It is still too early (January 15, 1915) to hazard a guess as to the final outcome.—Ed.]

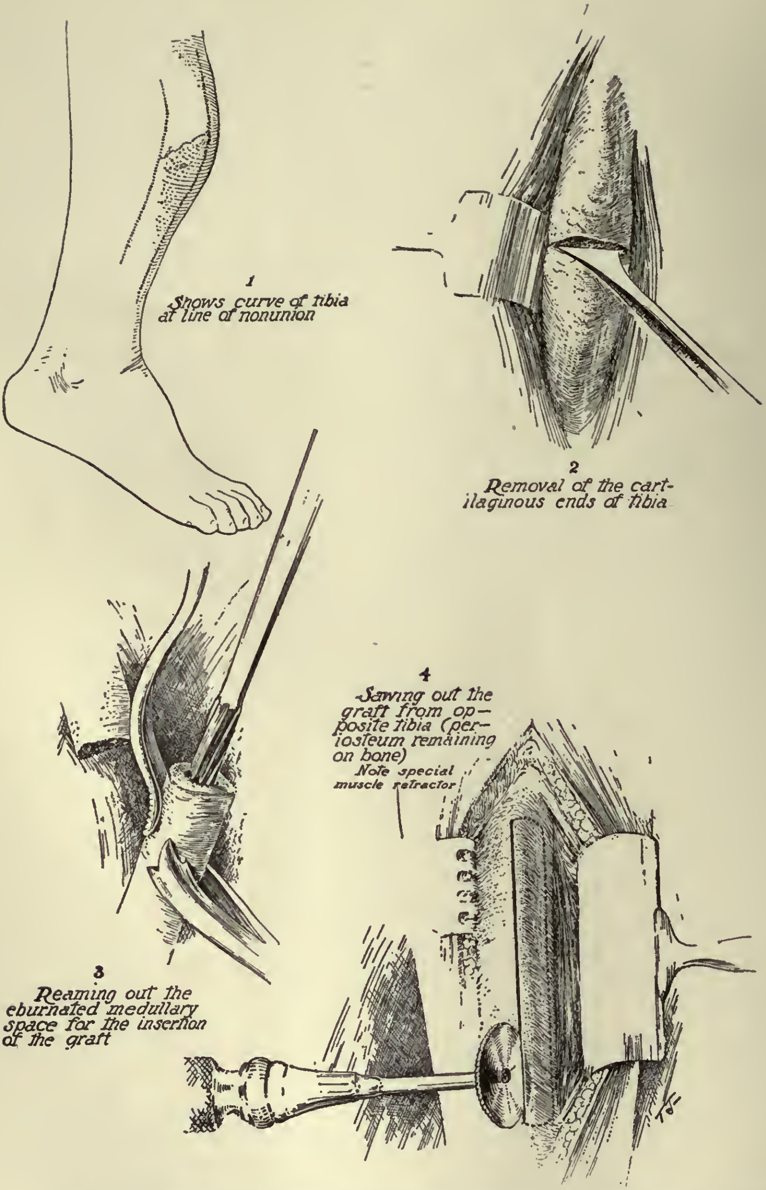
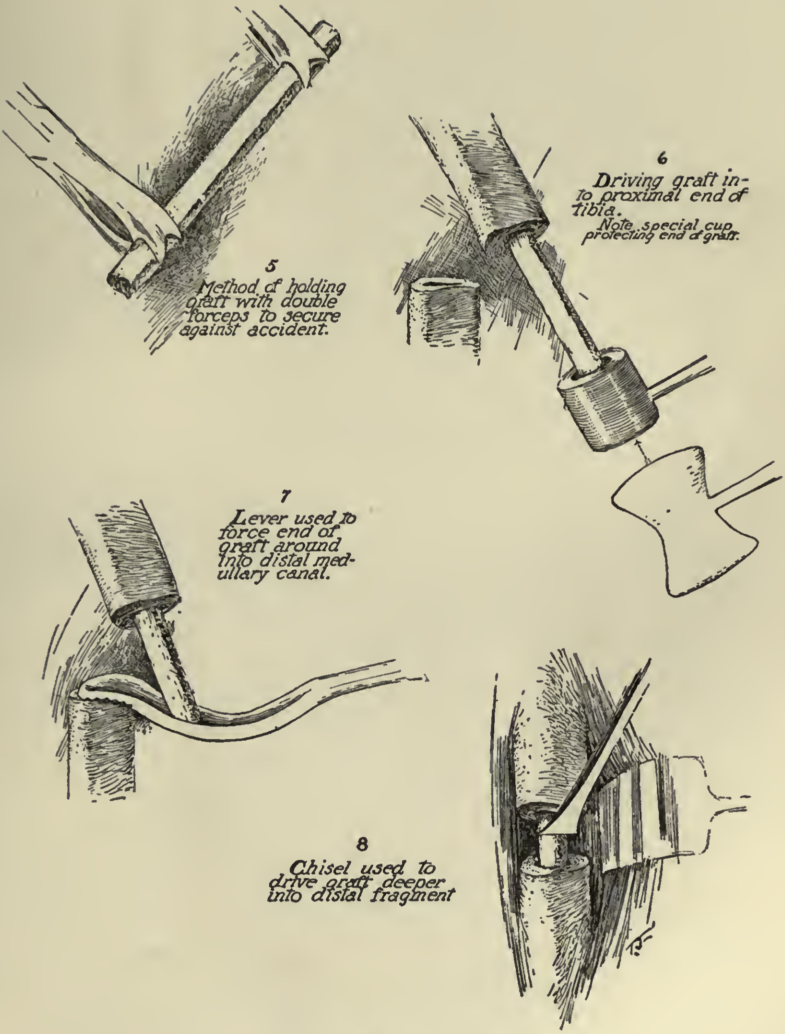

1
Shows curve of tibia at line of nonunion
2
Removal of the cartilaginous ends of tibia
3
Reaming out the eburnated medullary space for the insertion of the graft
4
Sawing out the graft from opposite tibia (periosteum remaining on bone)
Note special muscle retractor

Fig. 21 a.—A series of drawings illustrating Dr. Murphy's method of bone transplantation for non-union of the tibia, cases of which have already been reported in the Clinics. [In Fig. 2 the cartilaginous covering of the bone-ends has not been clearly outlined by the artist.—Ed.]



5 Method of holding graft with double forceps to secure against accident.

6 Driving graft into proximal end of tibia. Note special cup protecting end of graft.

7 Lever used to force end of graft around into distal medullary canal.

8 Chisel used to drive graft deeper into distal fragment

Fig. 21 b.

OPEN REDUCTION OF A POSTERIOR DISLOCATION OF THE SPINE AT THE LEVEL OF THE SECOND LUMBAR VERTEBRA—LAMINECTOMY

HISTORY

THE patient, a married man aged thirty-three years, entered the hospital with the following history: On August 24, 1914, the patient fell from a height of 15 feet, striking the ground, he thinks, on his back. He was not rendered unconscious by the accident. He tried to rise, but could not, for he found he had lost control of both legs. Sensation in the limbs was also lost up to the level of the great trochanters. The strength of the abdominal muscles appears to have remained normal. The patellar reflexes are absent. The Babinski reflexes are negative. He cannot flex or extend the legs. He cannot move the toes. The pricking of a pin is not felt in either leg below the trochanters. His left leg lies in slight external rotation. The range of motion in both hips on manipulation is normal. He has loss of control of both the sphincter ani and the sphincter vesicæ. In short, conductivity in the spinal cord appears to be lost below the level of the second lumbar vertebra.

COMMENTS AND OPERATION

DR. MURPHY (September 3, 1914): The patient has lost control of the bladder and does not know when his bowels are about to move. There is no voluntary motion in the lower extremities, and there is also complete absence of sensation below the line of the trochanters. The spinal lesion lies at the level of the second lumbar vertebra. Is that your estimate of the situation, Dr. Iseman?

DR. ISEMAN: Yes, doctor.

Dr. Murphy: I think the first thing to do is to look at the *x*-ray plate, because the *x*-ray gives us a very much better idea of the degree of spinal deformity in this case than we derive from an examination of the spinous processes. There is only a little deviation of the spinous processes demonstrable on palpation. When one feels this slight lumbar hump in the processes, one would say that there cannot be much displacement of the vertebral column, and that the cord, therefore, is not more bent or damaged than the concussion would damage it at this level. The lateral *x*-ray picture shows the posterior displacement in the upper half of the lumbar spine. One cannot see these *x*-ray details well from a distance. The line of dislocation lies between the first and second lumbar vertebræ. The posterior margin of the body of the first is on a line with the anterior portion of the body of the second lumbar. This radiogram was made before reduction was attempted, at a time when the visible spinal deformity was very marked, and represents the original degree of deformity. The present deformity, since reduction, is much less marked, and the *x*-ray plate more ambiguous. There appears to be only slight lateral displacement, but somewhat more anteroposterior deviation, the details of which are clear neither in the frontal nor oblique pictures. In attempting to reduce the dislocation immediately after the accident two men made extension on the legs, two made counterextension on the shoulders, and Dr. Iseman made direct pressure over the dislocated vertebræ. The displaced lumbar vertebræ slipped forward with a snap; but the deformity, while much decreased, was not entirely overcome, and all further efforts at reduction were of no avail.

What shall we do with the case now? In the effort at the reduction it was at first believed that the displaced vertebræ had been restored to their normal position. Only the slight remaining deformity spoke against that view. The *x*-ray pictures of yesterday and of the day before perhaps rather justify us in believing that this may be true, and that the absence of any improvement in the cauda equina symptoms may be due not to continued pressure on the cauda, but to a transverse lesion of it

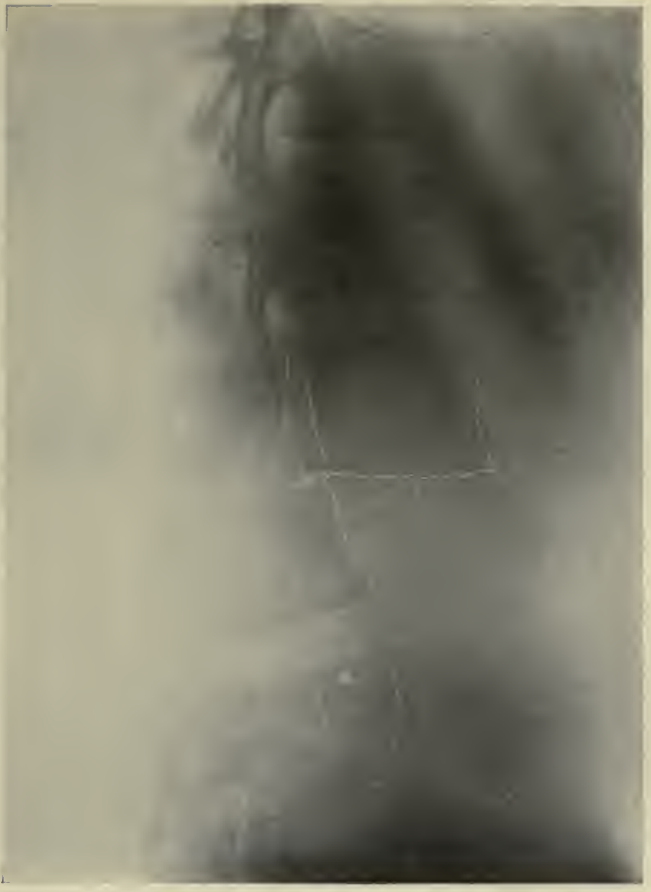


Fig. 22.—Posterior dislocation of the spine at the level of the second lumbar vertebra, the result of a fall on the back. Lateral roentgenogram made immediately after the injury and before any attempt at reduction was undertaken. Note the extreme backward luxation of the lower portion of the spine, the anterior border of the second lumbar vertebra lying in line with the posterior border of the body of the first lumbar. Note the angle of tilting—about 15 degrees—between the articular surfaces of the bodies of the two vertebræ—the first and second lumbar. It is only in the lumbar and upper cervical regions of the spine that so great a degree of dislocation can occur without an accompanying fracture. The white lines scratched on the plate outline the bodies of the first and second lumbar vertebræ.

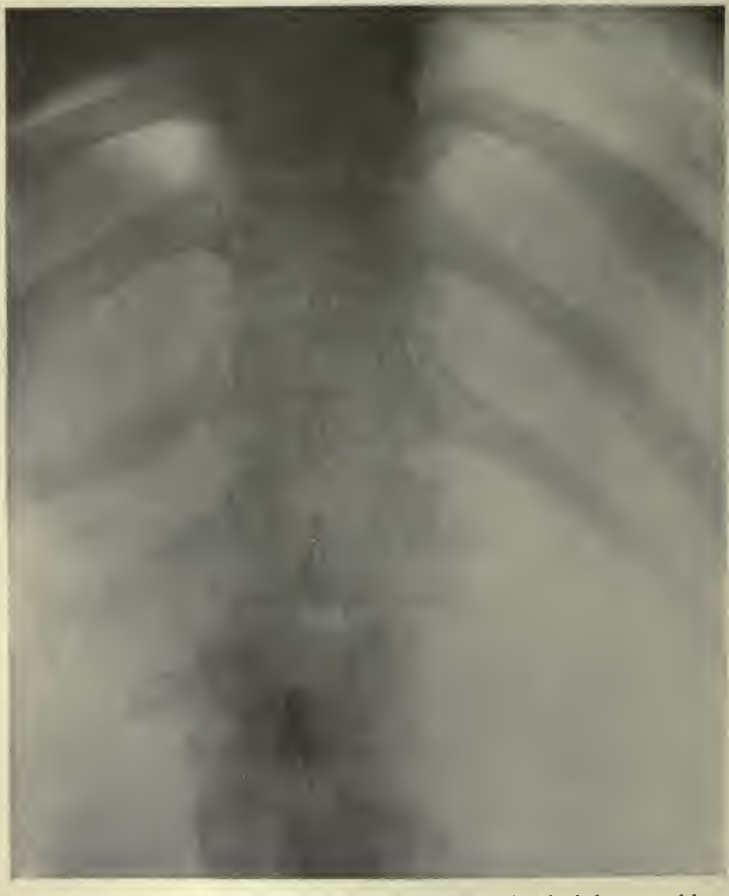


Fig. 23.—Posterior dislocation of the spine at the level of the second lumbar vertebra, the result of a fall on the back. Anteroposterior roentgenogram made after partial reduction of the dislocation was accomplished by extension and manipulation. Note the slight left lateral deviation of the second lumbar and the following vertebræ, and the absence of fracture.

produced at the time of injury. Remember that following the injury the paralysis of the lower extremities was *complete*, *circular*, and *instantaneous*.

In the monograph which we wrote on the subject of the surgery of the spinal cord and peripheral nervous system, which we published in 1906 in *Surgery, Gynecology and Obstetrics*, we made the statement that wherever that syndrome occurred in a lesion of the spinal cord it was useless to operate; but that statement concerned, however, the cord *above* the conus, not *below* the conus. Do you grasp the difference? In the spinal cord above the conus the axons have no neurilemma. In other words, the axons of the cord are aneurilemmic, and, therefore, incapable of regeneration. Below the conus the axon possesses a neurilemma coat just as do the peripheral nerves. Therefore their function should be restored if the caudal filaments are properly reunited, and we are, accordingly, going to expose the cauda to-day and examine the condition of its filaments. If we find them divided, and if we can locate the corresponding ends, we wish to bring them together and suture them, thus putting them under the same favorable conditions for union that we should if we had a divided peripheral nerve to deal with. We know that a peripheral nerve, when divided, always has its function ultimately restored if properly approximated. The same statement holds true for divided caudal filaments.

The older men present, and the men who have looked over the literature, will remember that this was not always the accepted view of neurologic surgeons. You will find that the literature of this subject divides the clinical observations into two distinct classes of cases—the failures and the successes following nerve suture. One group of men state that the suture of a nerve is not followed by a reëstablishment of function, because their cases were not successes. By the way, Moses Gunn, former professor of Surgery at Rush College, was one of the first operators to publish the report of a successful nerve anastomosis. He made a reapproximation of the divided musculospiral nerve with subsequent reëstablishment of function. That was away back in the days when I was a student at Rush.

Why have we this controversy in the literature, and why were the surgeons divided into these two distinct schools? As I analyzed, in 1906, the cases from the literature and from my observation, I came to the conclusion that the schism was due to the fact that one group of surgeons had sutured the upper nerve-end, the enlarged bulb, without freshening its distal end, whereas the other group of surgeons *had* freshened it. If one does not freshen both the nerve-ends by cutting off the end-bulbs, so that one can see the brush-like protrusions of the nerve fasciculi sticking out of the cut nerve-end, union and function are no more likely to be restored than if one attempts to unite two pieces of bone which are both covered over with a fibrous capsule. The proposition is just the same. The uncovered axons must come in contact with one another without the interposition of any connective tissue if union is to ensue and function be reëstablished. If this intimate and accurate contact be secured, reëstablishment of function may obtain as late as twenty-six years after division has occurred. We have one case successful after just that length of time. Complete regeneration occurs even when the nerve is sutured years and years after the dividing injury, *if the nerve is properly sutured.* "Properly sutured" includes getting the axons exposed for contacting, as well as securing the exposed axons by suture in accurate and close apposition with one another. Regeneration is established in the peripheral distribution of the nerve by the continuation of the growth of the axis-cylinders from the central side of the divided nerve in a centrifugal direction through the old axonal neurilemma sheaths.

Since the man's injury lies in the caudal zone and not in the true cord, and since the axons in the caudal zone have a neurilemma sheath, we are going to operate with the intention of reuniting the caudal fasciculi if we find them divided. If the injury had been above the conus, we should not consider an operation of any possible value to this patient with his complete transverse lesion.

How much deformity of the spinous processes was present at the time when you first saw the patient, Dr. Iseman? Was the displacement lateral or anteroposterior?

Dr. Iseman: It was purely anteroposterior.

Dr. Murphy: It appeared then just as it does in the first x-ray plate. The second lumbar vertebra was luxated backward on the first. The more recent roentgenograms apparently show that there is a backward luxation of the second vertebra still present, but it is only a slight backward luxation, not the severe luxation shown in the first x-ray picture. This spinal irregularity is just a slight kyphosis, rather than the deformity of a severe luxation. Before reduction the prominence formed by the dislocated second lumbar vertebra stuck far out. The reduction, I should judge from the appearance of the deformity, has been carried as far as continued pressure and extension can carry it. Looking at the patient's back, one sees that the posterior deviation of the spinous processes is very slight at present. The "knuckle" in the lumbar column just now is not over ⅓ inch in elevation above the plane of the back, and there is practically no lateral displacement—not more than ¼ inch. How many days has it been since the accident?

Intern: Ten days.

[Dr. Murphy made the usual median longitudinal laminectomy incision.]

Dr. Murphy: Here is a hematoma just beneath the skin. Here is also a laceration of the ligaments which hold the spinous processes together. This is quite the most difficult region in the spine in which to perform a laminectomy, because the spinous processes are so long and the transverse processes lie so deep.

[Dr. Murphy dissected down upon the transverse processes of the posteriorly dislocated second lumbar vertebra.]

Here is a projection of bone which I am unable to identify at once. Its internal surface looks like the facet of the transverse process. I first thought it was the stump of a spinous process dislocated to one side. It points backward and laterally. The same type of process exists on the other side. They *are* the transverse processes. I am taking the same precaution with this dislocation that we do with all our fracture and dislocation cases—waiting for ten days after its occurrence before performing an open operation. At that time the lymphatics and tissue spaces are so blocked that the risk of infection is minimal.

I shall be able to tell which vertebra it is which has been displaced by counting down from the last dorsal spinous process. Now I have it clear. It is the second lumbar vertebra, the superior articular processes of which we see here projecting backward. In the non-operative attempts at reduction which were carried out very shortly after the accident, and which were partly successful, the dislocation was reduced as far as the projection of the articular processes would permit. When the superior articular processes of the second lumbar locked against the rear of the inferior articular processes of the first lumbar, further non-operative reduction became impossible.

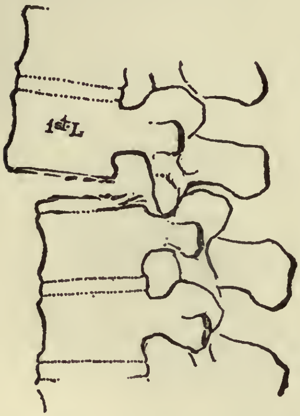


Fig. 24.—Posterior dislocation of the second lumbar vertebra on the first. Note the impingement of the superior articular processes of the second against the inferior articular processes of the first lumbar vertebra, which prevented complete reduction before operation. Note also the tearing of the interarticular disc between the two vertebræ, the increased interarticular space, and the slight angular deviation of the axes of the two portions of the vertebral column.

We have had the patient placed on the operating table flat on his abdomen with a pillow underneath him. By screwing up the gall-bladder elevator we can produce an acute flexion of his spine,—a kyphosis,—which will aid us in carrying out the reduction. If we cannot complete the reduction, we shall decompress the cord by removing the involved laminæ and spinous processes, "taking the roof off" the cord, as it were, and letting the cord free; but we prefer to reduce the dislocation, if possible. I am taking off the spinous process of the second lumbar vertebra because I feel that it will be of no special value to the patient here, and removing it will give me a much better view of and control of my field of operation.

[Maximum kyphosis plus extension failed to reduce the dislocation. Then Dr. Murphy had recourse to the use of the Lane bone skids, applying one on each side. The articular processes were thus unlocked with comparative ease and slipped by each other and into place with a snap. Dr. Murphy then performed a laminectomy in order to estimate the amount of damage to the cauda equina and to suture the fasciculi in case they should be found to have been severed or crushed by the pressure of the dislocated vertebræ.—Ed.]

This is better apposition than I had expected to obtain—practically a perfect apposition of the two portions of the vertebral column previously separated. The cord, or, I should say, the cauda, has an enormous amount of room now,—in fact, all its normal leeway,—and it can expand in any direction as freely as before the injury. It fills only a small percentage of the canal normally, as one can see here. I am freeing the cauda so that I can view it on all sides. I have opened the spinal canal for its full width before incising the dura and isolating the fasciculi of the cauda.

In incising the dura and opening the subdural space we are entering a zone of danger. One danger is the possibility of escape of an excessive amount of cerebrospinal fluid, and we easily prevent this by exerting a fair amount of firm compression on the unopened dura above the dural incision. The other and greater danger is that of infection, the only means of combating which is the routine practice of a rigidly scrupulous asepsis.

There is no ependymal canal in this zone—only the solid fasciculi of the cauda are present here. The apparent bulging of the cord here is due to the marked kyphosis which we have produced by the use of the gall-bladder elevator, which pushes upward against the patient's abdomen and flexes his trunk and spine.

Let the record show that there is complete suspension of faradic transmission below the point of trauma to the cauda, but that there is no apparent division or crushing of the fasciculi; therefore suturing is unnecessary.

Let the record show that the faradic stimulation of the nerve-trunks given off above the point of injury is followed by immediate muscular response.

I take it that we have to deal here with a traumatic suspension of function in the caudal filaments.

The facts of the case, however, do not completely support this view. If the axons were intact and their power to transmit impulses merely temporarily inhibited because of pressure, the axons below the point of pressure should transmit the faradic impulse. At least that is true when the pressure has been of short duration. When, however, the pressure has been of several days' duration, as may possibly have been the case here, since the dislocation was not completely reduced until today, that fact may alter the conditions of conductivity.

Let the record show that there was some blood found within the dura; that there is no evidence of a division or other lesion of the cauda lower down than the level of the dislocation.

VISITING DOCTOR: What is your prognosis?

DR. MURPHY: The prognosis is favorable, theoretically. I have had no experience exactly identical with this case, but, reasoning from other somewhat similar nerve injuries which I have had, I should rather expect to secure complete restoration of function here. I have no reason to presume that the axons have been cut, and if they have not been divided, there should be an early and complete restoration of function. If, however, there has been a break in continuity produced, it lies entirely within the neurilemma sheath, the separation is very slight between the two ends, and regeneration should be complete and fairly prompt. Of course, we know that, as a general rule, the closer an axon is cut to its cell-body, the longer will be the period of time required for regeneration to become completed and the greater will be the subsequent muscular atrophy. I have had, however, two cases of division of the brachial plexus where I sutured the cut ends of the trunks long after the original injuries and secured complete restoration of function in both cases. I hope for and rather expect an equally good result here.

Let the record show that there was no organized clot present inside the dura in the zone of the trauma—by that I mean no coagulation had occurred.

[The usual suture of the dura with catgut and closure of the wound layer by layer were carried out.]

Let the record show that we succeeded in doing all this work without contacting the hands with the wound. We have thus minimized the possibility of infection. It is a difficult thing to carry out this technic in a case like this one, but we succeeded in doing it, nevertheless. It adds about 25 per cent. to the operating time.

Contrast this case with the spinal case of yesterday. The patient yesterday had a slow and gradual compression of the cord from a granuloma, a tuberculosis lying external to the cord. We relieved the compression by performing a laminectomy. We did not open the dura to test the faradic responses, as we did to-day, because we feared the production of a tuberculous leptomeningitis; but the patient moves his feet freely up and down to-day, just as the result of taking off the pressure, and we believe, therefore, that he will soon attain complete or nearly complete recovery of function. That was a paraplegia produced by slow pressure.

This patient of to-day suffered a dislocation and a complete suspension of transmission below the point of injury—a paraplegia due to *sudden* pressure. Whether there will be a Wallerian degeneration of the peripheral segment of these axons following this traumatic suspension of function we do not *know*, but we *believe not*. Why? Because in blunt traumas to the ulnar nerve, for instance, where it is so exposed behind the elbow to trauma, and in traumas to the musculospiral nerve without division, one may have a drop-wrist which lasts for two, three, or four weeks, and is then followed by complete restoration of function. That is a suspension of function from pressure or trauma.

In Bell's paralysis one may have a suspension of function in the facial muscles often five or six weeks in duration, and followed nearly always by complete restoration. That, too, may be a pressure lesion, although we do not know for certain what the exact mechanism is. If a suppuration involves the facial nerve fasciculi and the axons are destroyed, a permanent facial paralysis results. Similarly, if suppuration occurs in the region of the cauda, and the caudal filaments are involved in it and destroyed by it, as occasionally occurs with tuberculosis of the vertebræ, there results a permanent paralysis of the legs. Here, I take it, a suspension of function is all that has occurred.

It was my belief originally that faradic irritation of the motor nerve-roots or of the nerve-trunks below them would always produce a response where the blocking of the impulses in the cauda or cord was due to pressure and not to actual severing of the axons. It may be, however, that in the early days after compression there is complete inhibition of faradic irritability below the level of the pressure, just as there is a complete inhibition of the deep reflexes often. That faradic irritability of the axons below the point of pressure is present, even when compression lesions have lasted months and years, we know from repeated personal observations. But whether it is also present in ten days or twelve days after the injury we do not know, because we never have had the opportunity of observing a case before so early after the original trauma since we have been using the faradic reaction as a routine test. Our emergency service is very limited at the Mercy Hospital. We know that in injuries to the spine of the mild pressure type one can have a complete absence of the deep leg reflexes, particularly when the injury lies high up in the cord, and these reflexes may remain absent for weeks and months, but, as a rule, finally return. It is quite evident that in such cases the vitality of the axons of the cord and of the peripheral nerves below the point of pressure has not been impaired, although their function has been temporarily inhibited. Of course, the great majority of patients who have a paraplegia after an injury to the spine, and accompanying that injury absence of the deep reflexes, are destined never to recover the use of their limbs, the absence of the deep reflexes being the manifestation of a complete transverse severing of the word.

Let the record show that the filaments of the cauda were not congested, thus differing entirely from the filaments of the cauda in the case of unilateral ascending neuritis due to sciatic nerve infection which we operated some weeks ago. (See CLINICS for August, 1914.)

A FURTHER REPORT

DR. MURPHY (September 5, 1914): We succeeded in getting this man to pass his urine by manipulating and massaging his prostate, and not by catheterizing him. He was catheterized once, however, by mistake, *before* the prostatic massage was tried. It happened this way: When I went through the ward on Sunday (August 30th), on my return home, I asked the intern how much trouble the patient was having with his bladder. The intern replied, "*No trouble at all. We have been catheterizing him.*" "Are you a graduate of Northwestern University?" I asked, and he answered, "No." I said, "If you were, I think I should have to have your diploma revoked, for if a student has attended my clinics and has not learned to keep the catheter away from such patients he has either a complete inability to learn or is totally deaf." Either defect disqualifies a man for the practice of our profession.

The early passing of the catheter is one of the things which is commonly done for these patients, and is one of the worst things that can happen to them. The patient must not be catheterized at all. If one catheterizes him a first time, the probability is that catheterization will have to be kept up, since a saprophytic cystitis is soon established and then catheterization becomes indispensable in order to relieve the retention and keep the kidneys from developing suppuration by the ascending route. We were more fortunate than usual in being able to establish voluntary urination after catheterization had already been begun, probably because the injury was located low down. By merely massaging the prostate we succeeded in getting the urine to flow. One can almost always start spontaneous urination in the beginning of a transverse cord lesion by *just a little* prostatic massage. Remember that this massage must not be too vigorous or severe, because one can massage the prostate so hard that a rectal ulcer is produced from the abrasions and the pressure.

My surmise regarding the cause of our failure to obtain a faradic response from the caudal filaments below the level of the injury has proved to be correct. I was inclined to believe that the absence of faradic response was a functional condition similar to the temporary loss of deep reflexes which sometimes follows pressure on the cord. I was rather forced to this conclusion because our careful examination of the caudal filaments showed absolutely no damage to them. Last night he had twitching of the muscles of his legs. That has an entirely different significance from what it has with an injury above the conus. With an injury above the conus, occasional twitchings of the muscles of the legs may occur for years, even in spite of the presence of a complete paraplegia, because the cells in the anterior cornua are still living and can send out impulses if they receive them, although the connection with the higher centers is permanently interrupted. But when the injury is in the cauda, the twitchings indicate that the cell-bodies are still able to transmit impulses over the axons to the muscles, and that the axons are structurally intact. This patient's legs almost flung him out of bed last night. That settles the prognosis. Of course, the axons are still living and already have undergone slight restoration of function. That is a very gratifying surprise for me this morning.

The patient's general condition is highly satisfactory. His pulse and temperature are normal. They were not altered even by that extensive operation. I think that this favorable result is due largely to the fact that there was no hand contacting with the wound. Lane is the man who brought out the importance of avoiding hand contact in aseptic surgery, and accentuated it more strongly than anybody else. At first he did not know why he had such good results; but on looking over his cases he noted that the very successful results in bone work occurred in the absence of hand contact with the wound.

During all this operation neither the assistants nor I contacted a gloved hand with the wound, or with any surface which was brought in contact with the wound. Our hands touched only the handles of the instruments. The ligatures and sponges were not touched by the hands at all. It is not easy to develop the reflex arcs necessary for this work when one has been in the habit of using his hands for everything, as in the surgery of the abdomen.

The other spinal case—laminectomy to remove the pressure of a tuberculoma on the cord—has not had an untoward symptom. The motion in his legs is very good.

[Note.—December 19, 1914: The patient's trophic changes have markedly improved since the operation, but his motor functions are still feeble, although already slightly improved, particularly in the muscles of the thighs. He has had no cystitis and no painful distention of his bladder.—Ed.]

OLD COMPOUND FRACTURE OF THE RIGHT MALAR BONE RESULTING IN LOSS OF THE EXTERNAL WALL OF THE ORBIT.—OUTWARD DISLOCATION OF THE EYEBALL.—UNSUCCESSFUL PARAFFIN INJECTION.—SUCCESSFUL BONE TRANSPLANTATION

HISTORY

The patient, a young woman aged twenty-one years, entered the hospital October 26, 1914, with the following history:

Eighteen years ago the patient was kicked in the right side of the face, near the eye, by a horse. One hour later she became unconscious and remained so for two hours. When she regained consciousness she was rational. The pain was very severe at the site of the injury, and pieces of bone around the orbit stuck through the skin. There was a severe laceration just under the eye and the eyeball seemed to protrude. The parts became very much swollen and black, and remained so for five weeks. The right eye swelled completely shut during this period. The loose pieces of projecting bone were removed at once, and a drain inserted. During all this time the pain was intense and pus discharged freely. Chloroform was administered every time the eye was dressed during the first three weeks. She had no chills, but she does not know as to fever.

The swelling finally subsided, and the eye then showed an entropion (enophthalmos). The sight was not entirely destroyed, but was greatly impaired. She has had much difficulty in keeping the eye open and the lid continually droops.

Six years ago the pain again became severe and the intra-ocular pressure seemed intense. Four years ago a section of the upper lid was removed to widen the palpebral fissure. In April, 1913, paraffin was injected below and to the outer side of the orbit, with the apparent intention of supporting the drooping eyeball and relieving the unsightliness of the deformity. This injection relieved the pain partially for a time, but the latter has gradually returned and now seems particularly severe within the eyeball. The patient is extremely nervous and depressed on account of the long-lasting pain, the loss of vision, and the continued drooping of the lid. The eyeball is luxated somewhat outward and downward.

COMMENTS AND OPERATION

Dr. Murphy (October 27, 1914): The eye showed an entropion?

Intern: It seemed to sink in.

Dr. Murphy: You mean an enophthalmos. When you speak of an entropion of the eye, you mean a turning-in of the eyelid. Which lid drooped?

Intern: The right upper lid.

Dr. Murphy: Write it down so.

Dr. Murphy: The patient is extremely nervous and apprehensive for two reasons: First, she has severe pain, and, second, she has practically no vision in the right eye. The right eye has dropped downward and outward and is so displaced that it does not lie in the same plane as the opposite eye. This deformity led us to consider closely what happened in the original accident. In looking over the bony framework of the orbit we find that the orbital margin has been fractured at about the junction of the malar with the frontal bone, and the bony fragments which protruded through the skin and were removed after the injury, as the history relates, constituted practically all the orbital portion of the malar bone. When you look at this skull, which Dr. Tivnen has very kindly brought in, you see how surprisingly much of the malar bone goes to make up the floor of the orbital cavity.

This defect in the orbital wall and floor has permitted the eye to drop outward and downward for a distance of about ⅓ or ½ inch. This dropping is enough to throw the eye out of plumb, and would interfere with stereoscopic vision, even if the eye were not so nearly blind as it is. It is this lacking portion of the malar bone which we have to replace by our operation.

It is surprising that this relatively large malar defect does not disfigure the face more materially. One can scarcely notice the malar defect in the facial contour, although the *x*-ray and the dropping of the eyeball indicate how extensive it is. The paraffin injection, about which the history tells us, is probably responsible for the filling out of the facial defect, although it has not been able to remedy the dislocation of the eyeball. It is this ocular deformity for which she now seeks relief, rather than the pain from which she sometimes suffers.

The technic of this operation is not an easy matter to figure out. In addition to the injuries which we have already noted, the patient has suffered a division of some of the filaments of the facial nerve to the orbital region, and this injury has impaired the contraction of the upper half of the orbicular muscle. The lower half contracts perfectly apparently. She has had an operation, apparently a tarsectomy, performed for the drooping of the right eyelid. At any rate, the surgeon cut out a section of the upper lid. I have asked Dr. Tivnen to go over the ocular symptoms in the case, particularly vision. The patient sees very little with the dislocated right eye, although she is not completely blind in it.

DR. RICHARD J. TIVNEN: The ocular findings in this case are as follows:

VISION: R. E.: $\frac{5}{200}$
L. E.: $\frac{20}{24.6}$

RIGHT EYE.—*Lids.*—Slight ptosis of the upper lid; several nodules of paraffin are palpable in the subcutaneous tissue of the outer third of the lower lid and at the outer canthus; the palpebral aperture is narrowed; the function of the orbicularis muscle is slightly impaired, causing some difficulty in proper closure of lids.

Globe.—Slight exophthalmos; the globe is depressed ¼ inch and deviated slightly outward and downward; the *excursions* are relatively normal; the cornea and sclera appear normal.

Pupil.—Slightly larger than the left. *Reactions:* Direct, consensual, accommodation, and convergence are normal.

Fundus.—The nerve head is atrophic (temporal half); there is a hemorrhagic spot in the macular region.

Orbit.—The outer third of the infra-orbital margin is roughened and presents a "gap" or "cleft" at least ½ inch in width just below the region of the frontomalar articulation. The superior nasal margin of the orbit appears slightly depressed.

LEFT EYE is negative.

The history of the case as just read discloses that the injury sustained by the patient at the age of three years caused a severe laceration and contusion of the peri-orbital soft tissues and produced also a compound fracture of the orbit, involving the outer and infra-orbital margin, the superior margin, and the outer floor. Suppuration ensued and several pieces of bone were removed; the globe was contused, hemorrhage into the orbit ensued, and there resulted a retrobulbar injury to the optic nerve and apparently a macular hemorrhage. As a consequence of these traumata two distinct ocular injuries were inflicted: *first*, the normal support of the globe was disturbed and the present faulty position of the globe resulted; *second*, a macular hemorrhage and atrophy of optic nerve were produced which resulted in serious visual impairment. It was difficult to keep the right eye open because the upper lid drooped.

The patient experienced no other particular disturbance following the injury until about twelve years later, when she began to suffer with severe headaches, referred to the supra-orbital and frontal regions of both sides. These headaches have been present almost constantly ever since.

About five years ago she had two operations performed on the lids to increase the width of the palpebral aperture, with only slight improvement as the result.

In April, 1913, paraffin was injected "in and around the lower lid." Suppuration resulted and some of the paraffin had to be removed. This paraffin temporarily relieved the pain of which she had been complaining.

The patient now seeks relief for:

First, the correction of the faulty position of the globe, which, from a cosmetic standpoint, is a matter of considerable distress to her.

Second, for the severe headaches.

Fig. 25 shows the location of the orbital fracture. This fracture, as Dr. Murphy has stated, involves principally the frontomalar portion of the orbital wall and the adjacent anterior portion of the orbital floor.

Dr. Murphy's plan to repair the "gap" in the orbital margin and orbital floor by transplanting a piece of bone from the tibia of the patient is, so far as I am aware, the first operation of the kind performed for an injury of this character. Previously our methods for the correction of these deformities were limited to efforts to replace the fragments by wiring or nailing, or the usual attempts to obtain a suitable reposition. The "bone transplantation" method, as advocated by Dr. Murphy, is an original and valuable addition to our therapeutic armamentarium in such cases. In connection with this injury it is apropos to emphasize the fact that the damage inflicted on intra-ocular structures in injuries of this character is often very great, and is frequently unsuspected at the time of the injury, owing to the fact that the eyeball itself exhibits neither rupture nor other evidences suggestive of severe injury.

Injuries to the optic nerve, followed by optic atrophy and accompanied by retinal hemorrhages, both of which occurred in the present case, are frequently met with in this class of injuries. In addition, detachment of the retina, dislocation of the lens, and ruptures of the iris are quite commonly encountered. It is well to emphasize, therefore, that in all orbital injuries of this character an ophthalmoscopic examination of the injured eye should be carried out as a routine procedure.

Dr. Murphy: In making my incision, I am going through the old scar, so as not to involve any additional filaments of the facial nerve. We have in this location filaments from two nerves, the facial and the trifacial. The facial is the all-important motor nerve of the face. The trifacial is chiefly the sensory nerve, although it must not be forgotten that it also contains the motor nerve-supply to the masseter muscle.

[Makes the skin incision and continues the dissection downward.]

In exposing this field, I am stretching these few nerve filaments still remaining here rather than cutting them, because if one merely stretches a nerve, its function will return, whereas if one cuts it, its usefulness is probably finished. The paraffin in the field here obstructs my vision materially. It is bound up in a mass of cicatricial tissue, as is frequently the case when small amounts have lain a long time in the tissues. The connective-tissue strands have broken it up into small blocks, and this network of connective tissue and paraffin makes it difficult for me to get my bearings.

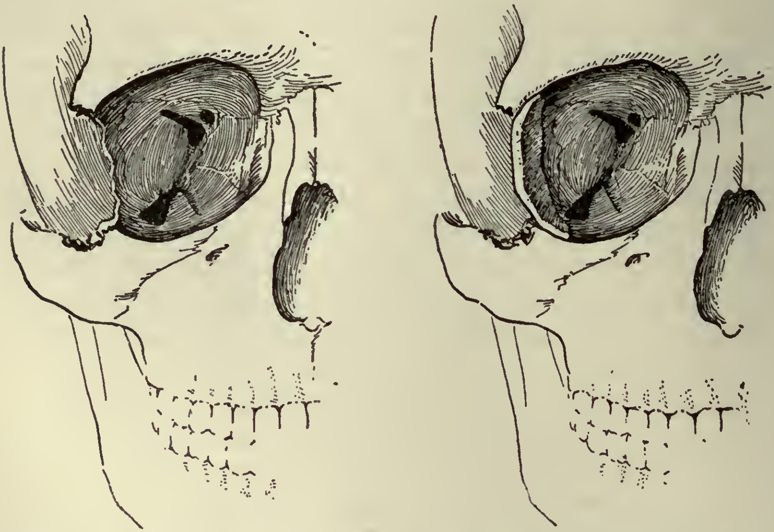


Fig. 25.—Old compound fracture of the right malar bone resulting in loss of the external wall of the orbit. Outward and downward dislocation of the eyeball. Unsuccessful paraffin injection. Successful bone transplantation. Note the large size of the defect in the malar portion of the orbital wall and the remodeling of the tibial fragment necessary to give it the curved contour required by its new position.

[Dr. Murphy continued dissecting away the paraffin. So hard was it that a portion was thought to be a remaining section of the orbital wall when palpated before operation; but in the course of the dissection its paraffin character was revealed. Another block made up part of the floor of the orbit. The space occupied by the paraffin mass made a large cavity when finally fully exposed.]

[After defining the extent of the cicatrix, which, by its contraction, was drawing the eye downward and outward, it was found impracticable to use the malar bone to fill the defect, because of the large incision which this operative procedure would necessitate, involving the probability of cutting the remaining facial filaments to the orbicularis muscle. If any of these important nerve-fibers had been cut in the course of the operation, the patient's eye troubles would have been as bad as, if not worse than, before operation. For that reason Dr. Murphy altered his original plan of shifting the malar bone inward to fill the orbital gap and transplanted instead into the orbital defect a suitable plate of bone from the patient's own tibia.]

[After thoroughly exposing the bony defect in the orbital cavity, the periosteum was dissected up from the inner orbital margin, and the bony margins freshened. The tibial transplant measured 1⅛ by ⅜ inches, and was ⅛ inch in thickness. The periosteum was not removed from its surface. The raw surface of the tibial transplant was placed in contact with the denuded area of the orbital margin, and between the latter and its stripped-up periosteum. The periosteum of the graft thus came to form the new inner lining of the orbital cavity, and the graft lies where formerly lay the malar portion of the orbit. The wound was then closed without drainage.—Ed.]

Dr. Murphy: The eye has now been elevated fully to its former level. One can no longer find any fault with the position of this eyeball, and we shall have no permanent trace of the deformity remaining except the slight white scar which she has had ever since the accident, and through which we operated.

Let the record show that we carried out this entire procedure without disturbing the eyeball at all, and that we produced but very little disturbance within the orbital cavity. The one hindrance to the work was the mass of paraffin present, which infiltrated all the tissues in our field of operation. It lay in some places in globules as large as a small bean.

When the bone transplant was placed inside the orbit, it did not have to be fixed in place. It merely rested there in the bed which we had prepared for it, and the pressure of the eyeball keeps it in perfect contact with the orbital margins with which it will soon develop a bony union.

The matter which concerns us now is purely the question of our asepsis. We had to touch this field of operation again and again with our gloved hands—much more, in fact, than we usually do in such operations, since we had so much difficulty in grubbing out the paraffin. But while we were preparing the tibial transplant, we had a sponge wet with 5 per cent. carbolic acid lying in the orbital wound, and we think that the carbolic acid will have taken care of any microörganisms which we may have introduced into the wound on our gloves.

[Note.—The wound healed by complete primary union. The bone remained in its position as fixed and held the eyeball in the proper relation to its orbit and the opposite eye.—Ed.]

UNUNITED BIRTH-FRACTURE OF THE CLAVICLE—ENDS FRESHENED AND UNITED WITH A LANE PLATE AFTER INVAGINATION

HISTORY

The patient, a boy aged nine years, entered the hospital with the following history: Three years ago his mother noticed for the first time some deformity of the acromion end of his right clavicle, which had appeared without any injury or other apparent cause. A doctor was consulted, who said that the condition was a broken clavicle, and had possibly existed from birth. The mother was in labor for eighteen hours at that time, and had a forceps delivery.

Examination at the present time shows a false point of motion at about the junction of the outer third with the inner two-thirds of the clavicle. There is cartilaginous crepitus here and considerable enlargement of the broken ends, apparently by callus. The drooping of the right shoulder and the clavicular prominence due to angulation of the fragments plus callus formation makes a readily noticeable deformity.

The patient does not complain of much, if any, pain or tenderness at the site of the fracture, and uses his right arm with surprisingly slight functional impairment.

COMMENTS AND OPERATION

Dr. Murphy (September 12, 1914): What was the presentation at birth?

Intern: I asked the mother, but she did not know.

Dr. Murphy: The record that you asked her is not here in the history, and it should be. A forceps delivery is probably a head presentation. But occasionally a breech presentation is delivered by forceps, and a breech presentation offers more opportunity for a birth-fracture of the clavicle than does a head presentation.

What was the first thing the mother noticed wrong with the boy?

Intern: She noticed on the left side of the clavicle a little protrusion which felt very hard, like bone. It was because of it that she consulted a physician.

Dr. Murphy: There *was* a protrusion in the lower triangle of the neck, just as the mother stated. It was particularly pronounced when the patient would bring the right shoulder inward, as by placing his right hand on the opposite shoulder. With the shoulder in this position the fragments sometimes pinched the skin in the lower triangle of the neck. Judging by the history, the physical examination, and *x*-ray plates, this is evidently an old, ununited fracture of the clavicle dating from birth. This boy's clavicle, from the tip of his acromion to his sternoclavicular articulation, is from 5/8 to 3/4 inch shorter on the side of the fracture than on the opposite side. There is some drooping of the right shoulder as a sequence. It has gone all these years practically unnoticed in an intelligent, well-to-do family. He has no pain and no trouble whatever from it, except the occasional pinching of the skin in the neck.

In planning our operative procedure here we must take into account the fact that we have to deal with a non-union of many years' duration, in a location where we are unable to produce perfect immobilization. If we make use of a bone graft—and this is a position where we should like to use a bone-graft to insure union—we have to face the insuperable objection that perfect immobilization is necessary for the healing of the transplant in place.

Since bone transplantation, therefore, is out of the question, we are forced to do that which we talk against and which Mr. Lane always decries—to use the Lane plate in an attempt to cure a non-union. Remember that, as a rule, a plate should not be used where there is a failure of union. That is just the opposite of the views of the early users of the plate. In the clavicle, because of its mobility, and on account of the youth of this boy and

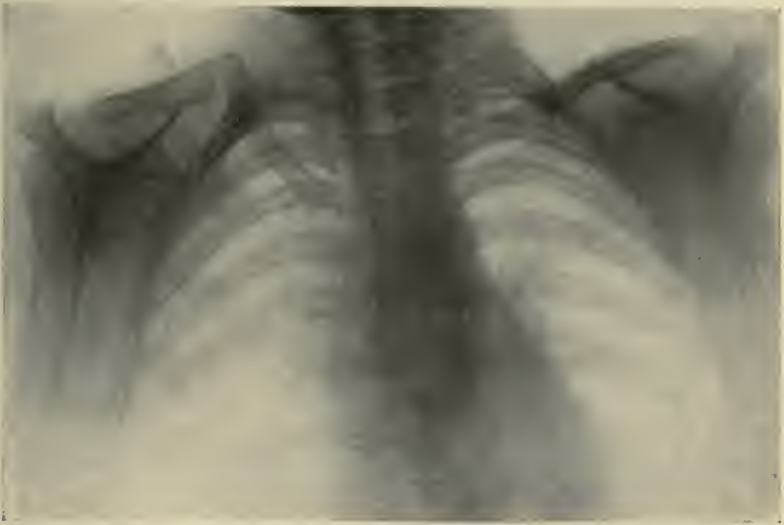


Fig. 26.—Ununited birth-fracture of the right clavicle. Note the sharp upward angular deformity, the pseudo-arthrosis development, the bulbous enlargement of the broken ends, and the drooping of the right shoulder. Note also the downward displacement of the right sternoclavicular articulation and the wide interarticular interval of the right acromioclavicular joint. Postero-anterior roentgenogram made before operation.

the difficulty of restraining his movements, I believe we can get a better and more accurate adjustment of the fragments and retain them in position better with the plate than in any other way; and, on account of his youth, we hope to get a union here, in spite of the bad reputation of this mode of treatment in these cases. If he were an adult, we should say that he would probably not get a union with the plate. If he were an adult we, therefore, should put in a bone transplant and a four-screw plate—the transplant to insure union and the plate to immobilize it and the fragments. You cannot secure the same immobilization with a child as with an adult. That is the chief reason why we are *not* using a graft and why we *are* using the plate in this particular case.

In non-union of bones there is no single hard and fast reason for the failure of osteogenesis. The most common and well-recognized cause of non-union in fractures of the long bones, such as the femur and tibia, is the interposition of non-osseous material, such as fascia, fat, or muscle. However, the cases are not at all infrequent in which this is not the case, and some other explanation must be sought. After fracture of the tibia and fibula sometimes the tibia unites, but the fibula does not. More commonly, perhaps, the fibula unites and the tibia fails to do so.

There are several definite sites in the long bones where non-union is particularly common. One of the most frequent is the junction of the upper with the middle third of the humerus. The next in the percentage table is the junction of the lower with the middle third of the tibia. Then comes the junction of the lower third with the middle third in the forearm, and then the lower third of the humerus and its condyles. The next in frequency is the junction of the middle third of the femur with the upper third.

In looking for an anatomic feature common to all these frequent sites of non-union one is struck by the fact that the bone structure is compact in all these locations, and that non-union rarely occurs in the cancellated areas of long bones. Callus seems to form more readily and bridge over the break in cancellated than in compact bone.

VISITING DOCTOR: Why do you prefer plating to wiring such a fracture?

Dr. Murphy: By wiring the fragments one can get them into apposition without difficulty, but one cannot keep them perfectly immobile during the process of healing, as is necessary in this case in order to secure union; but by using a four-screw Lane plate one can not only keep the fragments in apposition, but keep them practically free from motion on each other. The action of the pectoral muscles and the weight of the shoulder continually tend to pull the fragments out of line after they have been properly set. Therefore, very positive mechanical measures are necessary to maintain the alinement. Unless we make an autotransplant of bone, we must expect to get some shortening of this clavicle ultimately. But bone-grafting is out of the question here, as we have already stated. The shortening, however, will not be any greater than it is at present, because the little that we shall lose by freshening the ends of the bones will be fully compensated through eliminating the present angular deformity of the clavicle by straightening the bone.

[Dr. Murphy made the skin incision below the clavicle and parallel to it, in order to keep the scar away from the neck. A little dissection freed the site of the fracture and brought the pseudo-arthrosis in view. He then demonstrated it to the clinic.]

Note the development of this thick capsule around the pseudo-arthrosis. The strength of the capsule is equal to that of a normal joint of the same size, if it is not actually stronger. It is made up of connective tissue which has developed around the site of fracture to help hold the bones in apposition. The point of greatest difficulty in getting off the periosteum will be at the site of the pseudo-arthrosis. This bone has changed its contour to fit its new line of support to the shoulder, and it no longer presents its normal anatomic conformation.

[When Dr. Murphy opened the pseudo-arthrosis, a membrane closely resembling synovial membrane was found lining the end of each fragment and forming a complete new joint.]

This is not a true synovial membrane. The fluid in the joint is not a true synovial secretion; it is a collagen and, accordingly, non-absorbable.

[Dr. Murphy freshened and reshaped the ends of the two

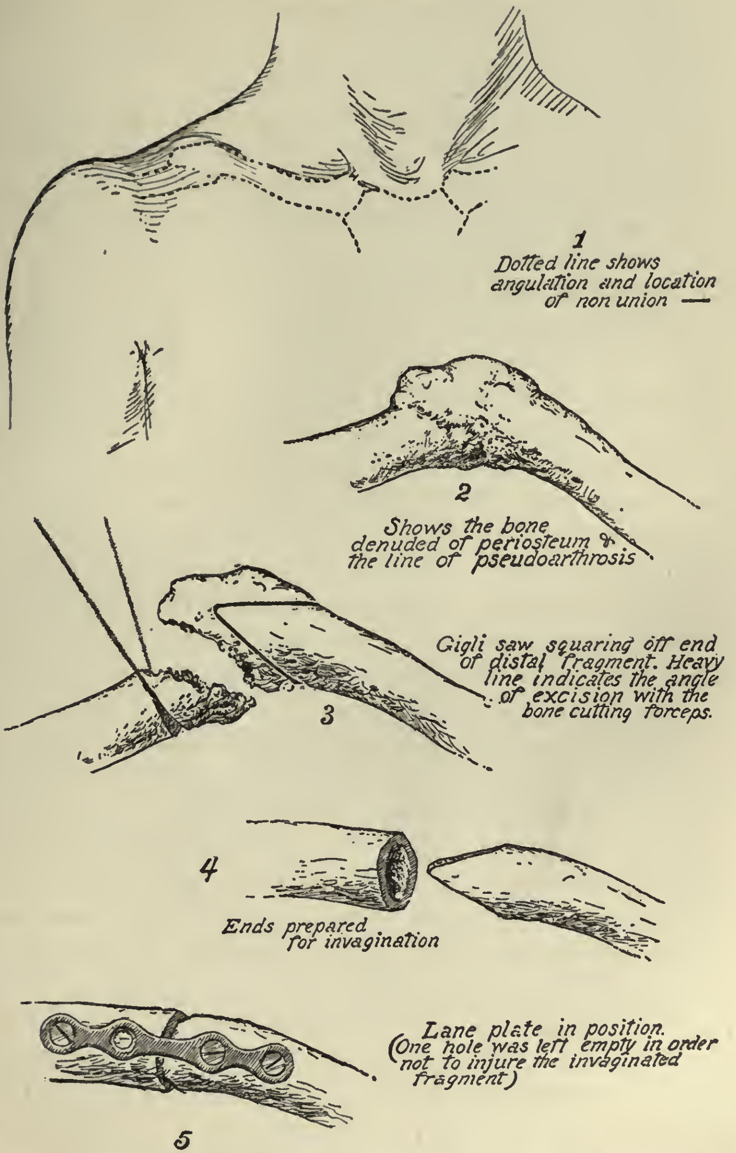


Fig. 27.—Ununited birth-fracture of the clavicle. Bone-ends freshened and united with a Lane plate after invagination.

fragments, as shown in the accompanying illustration (Fig. 27). The end of the distal fragment was cut off at right angles with the shaft of the clavicle by using the Gigli saw, and its marrow for a distance of ½ inch or so was cleaned out with the reamer. The end of the proximal fragment was trimmed to a point with the bone-cutting forceps. The pointed tip was invaginated into the reamed-out distal fragment, and the two fragments fastened firmly and immovably into place by using a small vanadium steel four-screw plate. One screw was not inserted, in order not to break the tip of the proximal fragment.—ED.]

This is a much more difficult proposition than the plating of a femur. There one can do practically no harm while manipulating the fragments and inserting the screws. It is an entirely different situation when one is working on the clavicle with delicate and important vessels and nerves beneath the field of operation. This plate will probably be taken out later, when union has become well established. I removed the periosteum from the ends of the fragments for a considerable distance. That is one of the peculiar but necessary factors in obtaining a union in such a case, dissecting up the periosteum, but not cutting it away. Thus a cap of callus is formed around the neighborhood of the desired union.

VISITING DOCTOR: How long will you immobilize the fragments?

DR. MURPHY: Six weeks. Nature provides these false joints, due to pathologic processes, with all the attributes of normal joints, endeavoring to do in a reparative way just what we try to do in our arthroplasties. Note how perfectly nature does it! I believe that the successful production of a new joint is entirely a matter of technic. If nature can do it successfully, as she often does, why cannot we do it by following nature's technic or even improving on it? We *can* do it, and we *do* it, not quite every day, but pretty nearly every day, and our results are becoming better and better continually, as we are becoming more familiar with the small details of technic necessary for success.

I had a letter last night from a woman in Texas on whom I performed an arthroplasty of the hip a number of months ago.

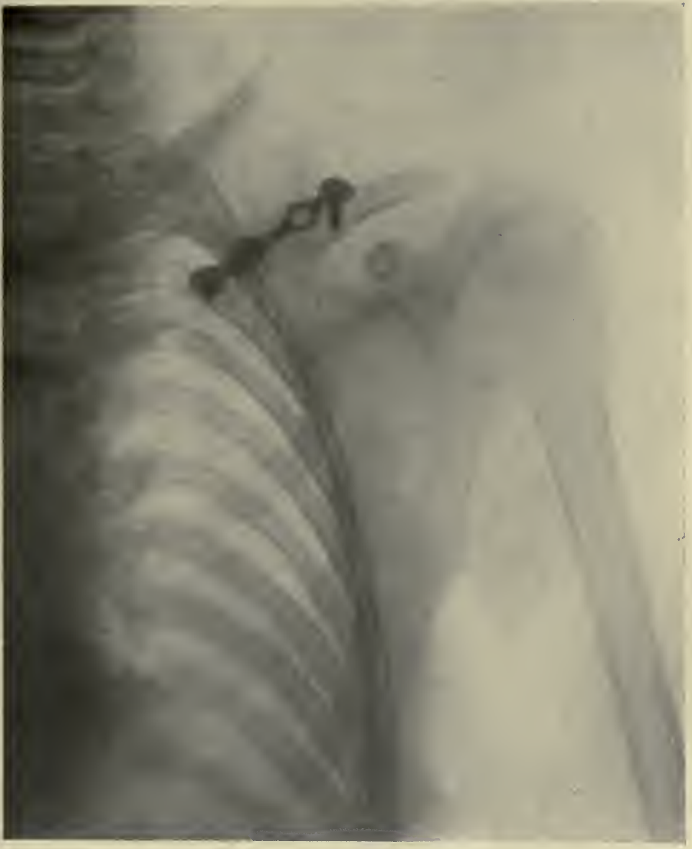


Fig. 28.—Ununited birth-fracture of the right clavicle. Broken ends freshened, invaginated, and fastened with a Lane four-screw plate, one screw being omitted in order not to injure the tip of the invaginated fragment. Roentgenogram made four weeks after the operation. Note the perfect alinement of the clavicle thus obtained. This plate was made with the patient lying on his back, whereas the plate before operation was made with the patient lying on his face; hence the apparent reversal of sides in the two roentgenograms.

She had had an ankylosis of the hip in an adducted position for five years, with great deformity and an unsightly limp. She writes that she is so well satisfied with the result that she would not be back in her old condition for the whole State of Texas; and that is a considerably larger tract of territory than the warring nations of Europe are contending for at the present time. She can walk upstairs with ease, and she writes that she "can do anything"; that is, as much as she could before her ankylosis developed. We do not get so beautiful a result every time. If we did, how happy we should be for the chance to arthroplasty the joints of Europe's crowned heads, and relieve their owners of their *Landlust!* In this particular case we made an unfavorable prognosis. We said she was so extremely stout that we were dubious about success, for we do not like to operate on stout patients. She takes pride now in telling us that fat people *do* get well after their arthroplasties. It is largely a matter of asepsis.

[The subcutaneous tissues were united with catgut to take all tension off the skin, which was closed with horsehair.]

We are sealing up the wound with celloidin instead of collodion, because the latter irritates and blisters a child's skin too easily. The patient will lie flat on his back in bed without a pillow or a back rest until the bone is completely reunited.

[NOTE.—The patient remained in bed on his back for four and a half weeks. The wound healed by complete primary union. The bony union and the position secured are perfect, as shown by the accompanying radiogram.—ED.]

CARBUNCLE OF THE ARM—SEPTICEMIA—METASTATIC PLEURISY—DEATH

HISTORY

The patient, a man aged twenty-seven years, entered the hospital July 5, 1914, with the following history: About two weeks ago a small crop of boils appeared on the patient's left shoulder, and rapidly grew to carbuncles. There were three carbuncles in all. Two of them opened of their own accord, and one was opened with a knife by a doctor on July 3, 1914. Pus drained from each one. On July 4th the third carbuncle was opened again by the doctor, and drained pus. The patient's temperature July 4th was 103° F. at the first reading, and, two hours later, 104° F. He had chilly sensations all that day, but no distinct chill. To-day (July 5th) he complains of much pain in the left side of the chest, and also some difficulty in breathing, the pain being especially marked on deep inspiration. He complains also of a dull, aching pain in his back. This pain runs down his spine to the level of the iliac crest. Late in the day yesterday the carbuncle was opened again, but no more pus was found.

COMMENTS AND OPERATION

Dr. Murphy (July 6, 1914): What was the leukocyte count yesterday?

Intern: 11,200.

Dr. Murphy: This is one of those dangerous spreading streptococcus infections deep in the fascia beginning as a carbuncle. The respiratory symptoms which the patient shows, taken in connection with his high fever and great prostration, even in the absence of definite findings on auscultation, incline us to the belief that this streptococcus infection has already extended along the deep lymphatics to the left pleura, spreading down, perhaps, to the pericardium also. This region of the shoulder is a bad place to develop one of these large carbuncles. It lies in close proximity to the deep set of lymphatics which passes upward out of the axilla, and it is, therefore, fearfully dangerous to life because of the great likelihood of rapid spread of the streptococcus infection along these wide-open pathways.

[Dr. Murphy made a deep crucial incision through the entire extent of the lesion and down to the deep fascia. The entire focus of pus-riddled tissue was turned into one large cavity by the use of a dull curet.]

There is where it (the carbuncle) is situated commonly. It extends clear down to the deep fascia covering the muscles, but did not penetrate into the deltoid bursa. See the foci of necrotic material as we break them up. See the pus welling out of the wound. Here is another deep pus pocket which I am emptying. [Completes the toilet of the wound.] This defect is not to be closed now, but will be left wide open with a loose packing of iodoform gauze, and allowed to drain. These silkworm-gut sutures are being put in for closure subsequently, but are now left long and will not be tied until the suppuration has been controlled.

Let the record show that the superficial tissues were all infiltrated with the products of inflammation clear down to the deep fascia covering the muscles, and that necrosis had extended through the skin into the subcutaneous tissues, involving both structures *en masse.*

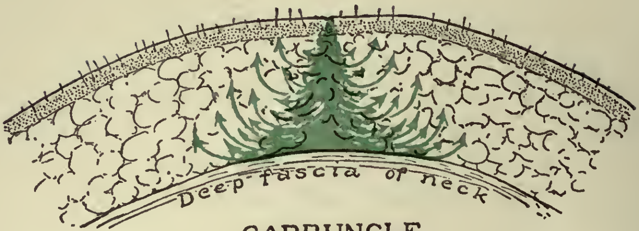
REPORT TO THE CLINIC NEXT DAY

DR. MURPHY (July 7, 1914): A patient who has a furunculosis of the upper lip always has a rather high temperature and a high leukocyte count. A furuncle on the upper lip is always a very serious lesion—enormously different in course and prognosis from what it would be on the arm or forearm or on the back of the neck. It always puts the patient in a dangerous situation. It may, in fact, be stated as a general truth that whenever a furuncle or a carbuncle occurs in a rich lymphatic zone, the patient's life becomes enormously endangered by it. A carbuncle, however, on the upper lip is, as a rule, fatal, and very promptly fatal, and this rule, too, may be generalized to the effect, namely, that a carbuncle in any zone richly supplied with lymphatics is, usually, fatal.

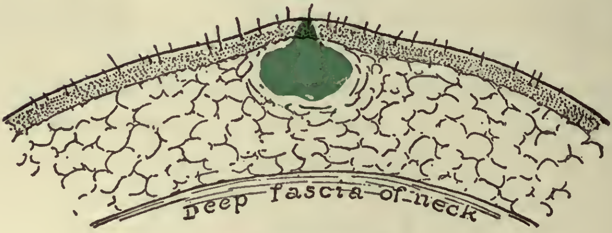
We have in the hospital now as a patient a young man who has developed recently a large carbuncle near the angle of his shoulder, on its anterior surface, extending into the axilla. Not only does this carbuncle lie in a zone relatively rich in lymphatics, and, therefore, dangerous to life, but it lies adjacent to a still richer lymphatic zone, the axilla. The carbuncle made its appearance on Tuesday or Wednesday of last week. The patient had something of a chill on Saturday (July 4th), rapidly followed by fever up to 103° or 104° F. That was a sign of the extension of the infection. Sunday he was breathing at the rate of from 50 to 60 respirations a minute, and his pulse-rate was 120 per minute. His temperature has ranged from 103° F. upward, but the striking symptom in the case has been his rapid, panting breathing. The method of his breathing, the fact that he breathes rapidly for a few seconds, then fixes his lower chest, breathing only with the upper portion of his chest, and then after another five or ten seconds starts his panting breathing again, is peculiar. He complained also of pain in his chest; then he complained of *severe* pain in his mediastinum; and, finally, he complained of severe periodic pain in the same location. We incised and evacuated the carbuncle yesterday morning, but without obtaining any material improvement in his general condition. The pain in the mediastinum is continuing, and his respirations last night were ranging between 56 and 60 per minute, his temperature remaining around 103° F. His pulse-rate this morning is 128. What does all that mean? Just exactly what it would have meant in the case of a carbuncle on the lip. Whenever you find that type of infection in a rich lymphatic zone, always look out for a further regional lymphatic spread or distant metastasis by way of the circulation. Such patients frequently develop a septic endocarditis. Then, either from the original focus, or from the endocardial vegetations, the patients rapidly develop metastatic foci of suppuration in other positions in the body. This boy has developed such metastatic processes in his diaphragm and pleura. That is why he breathes in that peculiar way. He holds his breath as long as he can. Then he takes a few short, quick respirations, and stops at the height of respiration with a sudden "catch" or grunt. Then he holds his breath again. And so the cycle is repeated. Remember, the diaphragm is the piston of respiration in the cylinder of the chest. The patients with a diaphragmatic pleurisy stop breathing as long as they can and, when forced to, make shallow, quick excursions, in order to put the diaphragm under the least possible tension and suspend pleural friction. The predominating feature in the clinical picture presented by this patient this morning and Sunday was the left-sided pleurisy, confined apparently at first to the upper diaphragmatic surface, and then gradually extending upward, particularly along the lateral wall. This is probably a metastatic pleurisy from which the patient is suffering. He may develop also a metastatic arthritis in one or more joints, a metastatic pericarditis, or a metastatic endocarditis. These conditions terminate fatally in a large percentage of the cases. If he develops metastatic foci in addition to his pleural infection, he will undoubtedly die.

I remember a very striking case of that type that I was called to take care of when I first started private practice. The patient was a west-sider, who had acquired a Neisserian infection of his urethra. He ran the usual course until the twentieth day, when he had a chill. Then he developed a pleurisy, a pericarditis, suppurations in his joints, and a suppurative peritonitis, all before he died. All these infections were metastatic from his urethritis. Such complications are not common with a urethritis, but at the same time they are usual enough. I cite that case merely to point out the common locations in which these metastatic infections appear, not only with urethritis, but also with furuncles and carbuncles, which arise in a richly supplied lymphatic zone.

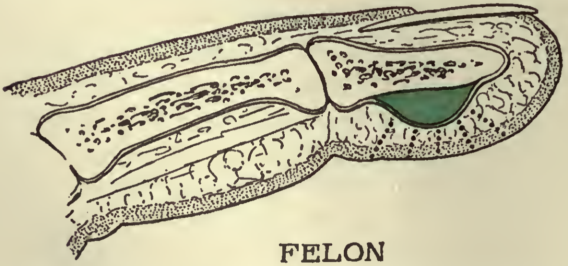
I wish, further, to call your attention to the serious results of defective treatment of a few comparatively minor surgical conditions, which are frequently inefficiently treated. Carbuncle is one of them. What do we mean by "inefficiently treated" in such cases? Making in the carbuncle a little slit which just goes through the derma is ill treatment. In a carbuncle a clean-cut incision should be made clear down to the fascia, and even beneath it, if necessary; then a transverse incision at right angles to the first cut, and then curetment of the entire necrotic focus or dissection upward of the flaps. The former method endangers the patient's life, whereas the second saves it. Always make the incision in a carbuncle deep enough to expose the underlying fascia. In opening a carbuncle remember that it is not a superficial infection. A furuncle is a superficial infection, but when it extends to form a carbuncle, it develops deep roots. These deep roots may be compared to the deep tap roots of the eucalyptus tree, which shoot downward until they strike rock and then spread like the banyan in every direction. Much the same course of events occurs in the development of a carbuncle, and that is what distinguishes it from a furuncle. The line of infection in a carbuncle extends downward in a relatively straight direction until it meets the deep fascia; then it spreads out along the surface of the fascia beneath the fat. That is why a superficial incision in the skin does no good. One does not reach the center of the infection. One must incise a carbuncle clear down to the fascia. One can easily trace the course of the necrosis down to the fascia, where it spreads out. To get rid of the necrotic focus and to give the patient an opportunity to get well, one must make the incision include the entire depth of the infected tract. The pathologic process in a carbuncle is not a lymphangitis: it is a necrosis. Necrosis occurs very early in the course of the inflammation, and the greater the delay in instituting proper treatment, the more extensive will the necrosis be. The route of infection is probably usually by way of a hair-follicle—since carbuncles occur almost exclusively in hairy regions. That it may occasionally occur by way of the sweat-glands also is a possibility which cannot well be denied. At any rate, once the hair-follicle or the sweat-gland is infected, the pus breaks loose into the surrounding columna adiposa, if it does not find a ready vent to the outside. The pent-up infection then rapidly descends along the



CARBUNCLE



FURUNCLE



FELON

Fig. 29.—The location and direction of spread of the pus in carbuncle, furuncle, and felon.

loose fatty tissue of the columna adiposa until it reaches the deep layer of the superficial fascia, which stops further downward progress and forces the infection to spread laterally, which it does with relative ease, the loose fatty tissue of the panniculus offering little resistance to its progress.

As the infection proceeds laterally it infects the roots of various columnæ adiposæ, and these secondary infections extend upward toward the skin, where they frequently point and make the multiple tiny pus points surrounding the central original focus of infection. Superficial necrosis occurs relatively early because the spreading deep phlegmon early cuts off the blood-supply to the fat, superficial fascia, and skin. The fat and fascia die early, but the epidermis, because of its great vitality, may live after the dermis and fat have been destroyed. The carbuncle continues to spread laterally until either an effective leukocyte wall is formed or appropriate treatment instituted. To drain a carbuncle properly the deep phlegmon must be opened widely by a crucial incision, extending laterally a little beyond the peripheral zone of infiltration, and clear down to the deep layer of the fascia. The four flaps thus made should then be dissected back to their bases with the knife or curet. Thus the whole area of the phlegmon is laid wide open and is packed loosely with moist or vaselinated or iodoform gauze to keep up free drainage. *Do not cut away the skin!* Let it fall back over the gauze. If it lives,—and it usually does,—subsequent skin-grafting becomes unnecessary.

The downward spread of the infection in a carbuncle is not unlike the downward spread of the infection of the finger-tip which results in the formation of a felon. An infection beneath the epidermis on the palmar surface of the fingers follows down the columnæ adiposæ to the periosteum of the phalanx, and then spreads along the bone until it is checked proximally by the joint-capsule, laterally by the attachments of the flexor profundus tendons, and distally by the skin of the finger-tip. Hence pus accumulates under pressure in this closed space, with what result? The blood-supply to the diaphysis of the phalanx is shut off, the periosteum and subcutaneous fat and connective tissue become necrotic, and the diaphysis of the phalanx dies and makes a sequestrum, which must ultimately be removed before healing will take place. The old idea of Roux that the lymphatics in the finger-tips run from the skin directly downward to the subperiosteal zone, and that a felon is due to the lymphatic transmission of infection to the subperiosteal zone by this lymphatic route, is no longer tenable. In the first place, we do not know that the lymphatics follow this route. In the second place, Sharpey's fibers run down into the osseous tissue here in such a way that it is practically impossible for the periosteum to be separated and differentiated as it is on other bones. In the third place, the proximal epiphysis of the phalanx is practically always spared, because its blood-supply is separated from that of the shaft and does not enter the closed space of the finger-ball. If a felon were a subperiosteal phlegmon, we should expect the whole bone to be destroyed. The pus which eventually forms beneath the periosteum is due to the presence of a sequestrum. Of course, besides this ischemia the necrobiotic and toxic effects of the infecting microörganisms play an important rôle in the production of the necrosis.

In opening a felon always make a lateral incision, so as to spare the nerves of sensation in the skin of the finger-tip, and make the incision down through the periosteum to let out the pus from the osteomyelitis focus beneath it. If the dead fragment has already separated, remove it at once, as well as the pool of pus in which it lies. It is remarkable how well the skin usually survives in these cases, and how relatively slight the disability in such a finger may be in spite of the shortening of the terminal phalanx due to the loss of the bone.

I learned the great desirability of the lateral incision for felon from a patient who was none other than Dr. Lee, with whom I was so long associated. Dr. Lee had one of his associates incise a felon, which was located on the doctor's right index-finger. The young man made an oblique palmar incision, knowing no better, and Dr. Lee himself knowing no better at the time. The incision, of course, divided the nerve-supply to the tip of the finger, and return of sensation never occurred, although the finger made an otherwise perfect recovery. Every time in later years that Dr. Lee threaded a needle or tried to carry out some delicate procedure with that index-finger he used to call down anathema on that unlucky assistant and on himself as well.

Seamstresses are an unfortunate class who not infrequently develop felons in the course of their work, and then have their earning capacity decreased by the similar incorrectly placed incisions of careless or inexperienced surgeons.

It is important not to confound a paronychia with a felon. The former commences on the dorsum of the finger, usually in the nail-fold, and spreads around the sides and base of the nail, and often underneath it. For its relief it needs the exposure of the root of the nail or the extraction of the nail, or both.

I spoke about carbuncle and the significance of it when it commences on the lip. I recall a man whom I visited some years ago in a suburb a short distance from Chicago. He had a carbuncle on his upper lip, and I had him come into Chicago to the hospital, although I said it was not anything serious, and that he would be all right in forty-eight hours. I was not so familiar then with the danger of this condition as I am now. We opened it up at the hospital promptly, but he was dead in thirty-eight hours as the result of a rapidly spreading streptococcus thrombophlebitis starting from it. That gives you some idea of the great danger latent in a lip carbuncle, and the speed with which it may, occasionally, prove fatal.

[NOTE.—No further metastases developed in the patient with the shoulder carbuncle. The streptococci were never isolated from the blood. The patient's toxemia rapidly increased, and he died on the evening of July 9, 1914, about forty-eight hours after operation. No autopsy.—ED.]

CONTRACTING CICATRICES ON INDEX-FINGER AND THUMB.—EXCISION.—PLASTIC OPERATION

HISTORY

The patient, a seven-year-old boy, entered the hospital with the following history: About six weeks ago he caught the right index-finger and thumb in a pulley. The flesh was torn off the finger and thumb, and the bone of the thumb was exposed. No stitches were taken. A dry dressing with powder was applied. The wound healed in about three weeks. The index-finger has developed its present flexion contracture in the past ten days. When the thumb is fully extended, it is painful. The index-finger is held flexed to a right angle. It can be flexed still further voluntarily, but cannot be extended voluntarily or passively because of the resistance of the scar tissue on the flexor surface.

COMMENTS AND OPERATION

Dr. Murphy (September 12, 1914): The patient caught his right hand in a pulley in such a way that the skin and flesh were torn from the anterior palmar surface of the index-finger, and, to a less extent, from the thumb. This is now one of the most unfortunate conditions which one can encounter in the hand, owing to the cicatricial contraction which so often takes place following lacerations or incisions of the palmar structures. The formation of an excessive amount of scar tissue after an injury in this region is not an infrequent occurrence. Whether the injury is a simple incision or whether it is a loss *en masse* of the tissue on the palmar surface of the hand seems to matter little oftentimes in regulating the amount of connective tissue thrown out. The excessive formation of connective tissue is quite a regular occurrence in anatomic locations, in which there is a free and unrestricted play of motion, almost exclusively flexion. These accumulations of callus keep constantly contracting and tend to produce limitation of motion, contractures, and deformities. This case is an instance of how rapidly marked deformity may ensue.

A similar occurrence often follows the common operation for webbed fingers. The web is divided by snipping it up the desired distance with the scissors, and then suturing the palmar cut edge to the dorsal edge. There is primary healing at the end of two weeks, and splendid freedom of the digits. Then the scar tissue begins to contract and, little by little, the web reforms and the fingers are drawn together again, and the condition of the hand becomes perhaps a little worse than before operation.

That is one way *not* to do an operation for webbed fingers. Another method which gives a result almost equally bad is that which makes use of a dorsal and a palmar flap, one wrapped around the raw surface on one finger, and the other around the raw surface on the other finger. The same gradual contraction of the scar also takes place here very frequently, but is a flexion contracture instead of a reformation of the web.

Neither of the tendons to the thumb is involved in the thumb cicatrix. The patient can still flex his index-finger at all the joints, showing that his flexor tendons are still functioning, although the flexor sublimis may be caught in the scar; but he cannot extend his finger because of the firm resistance of this dense cicatrix on the palmar surface.

The general plan of procedure which one follows in such a case is first to excise all the connective tissue, and then place a pedicled flap of normal elastic skin between the two wound edges. So long as the normal skin is there to stretch freely in flexion and extension, the formation of scar tissue will be minimal. This general procedure applies equally well to scars on all flexor surfaces, whether on the neck, in the fold of the wrist, the fold of the elbow, or the popliteal space. These are the common places in which a similar dense scar may form, because it is subjected here to frequently repeated extension and flexion.

The evil consequences of a scar in a freely movable area are, perhaps, most pronounced when the scar is circular and entirely

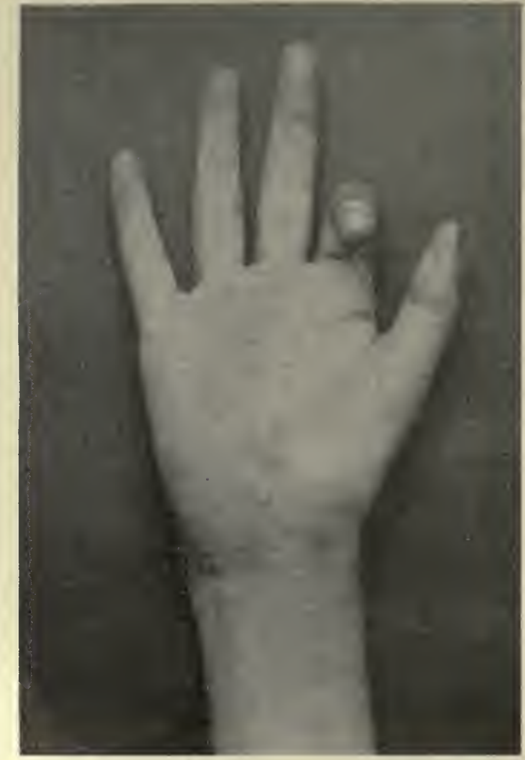


Fig. 30.—Contracting cicatrices on index-finger and thumb, the result of a tearing injury. Anterior view before operation, six weeks after the injury. Note the 90-degree flexion deformity of the index-finger and the relatively slight contracture of the thumb.

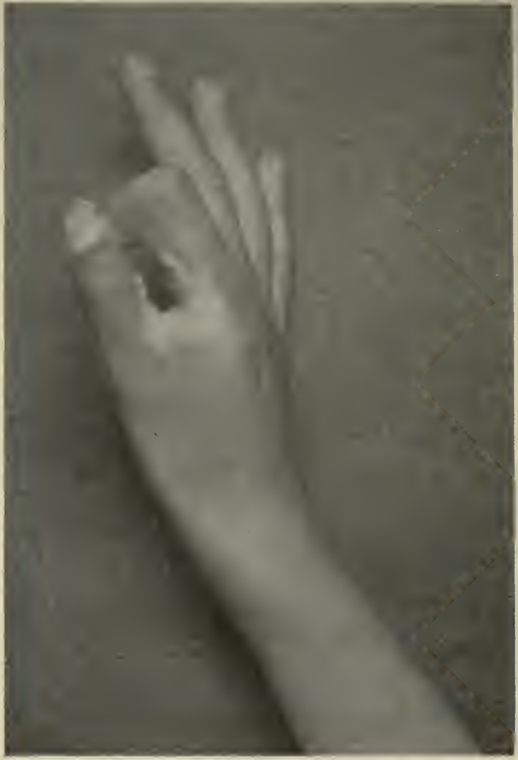


Fig. 31.—Contracting cicatrices on index-finger and thumb, the result of a tearing injury. External view before operation, six weeks after the injury. Note the extent of the white scar on the index-finger and on the interdigital fold, and also note the 90-degree flexion deformity of the index-finger which the scar has produced.

surrounds a limb or some natural orifice. The circular scar after the Whitehead operation for hemorrhoids may lead to stenosis and occlusion of the anus by exactly this same mechanism of excessive cicatricial deposit and subsequent contraction. In the Schede operation for varicose veins of the leg a complete circular incision is frequently made in order to interrupt all the superficial veins. This scar is continually subjected to the pull of flexion and extension during walking and running. A dense scar is frequently produced, which may continue to contract until it threatens the life of the limb by the severe constriction which it makes. I remember seeing in the County Hospital some years ago, when the Schede operation was in vogue, two cases in which there was an actual gangrene of the foot as a result of this cicatricial contraction. In the management of these Schede cases in the leg all one has to remember is the general rule that if the cicatricial circle is interrupted by inserting a small flap of elastic normal skin, further contraction will not take place or will be relatively slight.

I am first dissecting away all the scar tissue making up the cicatrix on the index-finger. Now that the dissection is completed it can be seen that the sublimis tendon of the index-finger was not involved in the scar. That fact improves the prognosis. We ought to secure a practically perfect result.

What shall we do next? I could draw both edges of the skin together and suture them, but it would not do the patient any good. In six weeks' time the scar tissue would all have reformed, and the finger would be contracted just as badly as it is now. What must we do? We must fill in the defect left from the scar excision by a slightly larger piece of normal, elastic skin. Such a piece can be secured by transplanting a pedicled flap from the front of the abdomen, since there is no place in the hand where we can take a skin-flap sufficient to fill in this gap without making an unsightly and awkward scar.

The next step is to free this contracted cicatrix on the thumb. It is not so serious at present as the scar on the index-finger, but as it contracts it will become more bothersome if not treated. Where this thumb scar runs back on the dorsum, it reaches a

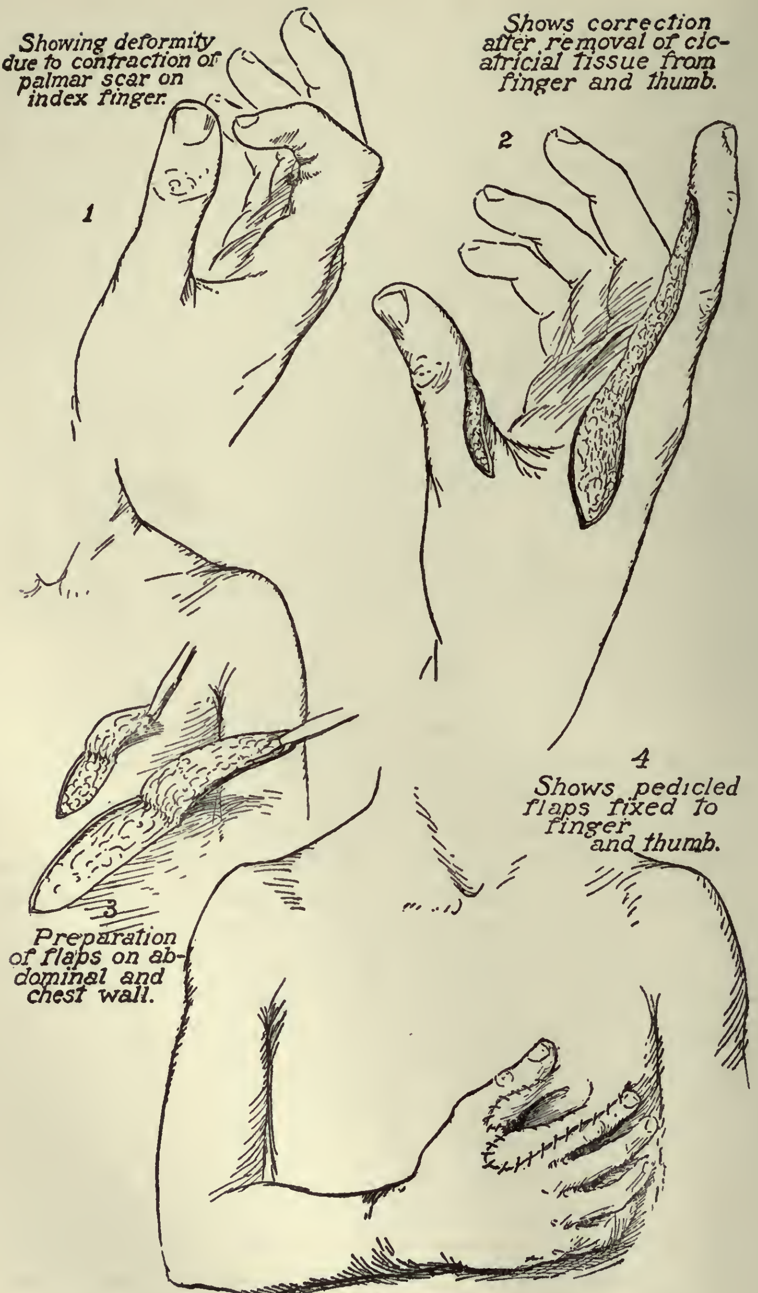

Showing deformity due to contraction of palmar scar on index finger.
1
Shows correction after removal of cicatricial tissue from finger and thumb.
2
3
Preparation of flaps on abdominal and chest wall.
4
Shows pedicled flaps fixed to finger and thumb.

Fig. 32.—Plastic operation for contracting cicatrices of index-finger.

zone where marked contraction is less likely to occur. We do not use iodin on the skin of these children in preparing them for operation, because iodin blisters occasionally and causes considerable complaint and an occasional infected wound.

I have dissected out the scar on the thumb (as shown in the accompanying illustration). It is not adherent to any of the important deep structures here. Now that it is removed, it would be even easier to bring together these two skin-edges than it was in the case of the index-finger; and the result of so doing would be equally unsatisfactory.

Our next step is to plan our incisions on the anterior surface of the abdomen, which has already been surgically prepared. (For the location of the incisions, see the accompanying illustrations, Fig. 32.)

I want to place the pedicled flaps so that the hand will lie in a comfortable position, for it must remain there until healing has taken place. Here we do not wish elasticity in the flap so much as sufficient skin. We do not take so large quantities of fat for transplants to the hand as we do where we are filling in other places. [Dissects up the abdominal flaps, sutures them to the defects on the hand, and then closes the abdominal wounds as far as possible toward the base of the flap.]

This is essentially an autogenous abdominal graft. We have made the flaps really larger than we need them, in order to assist in keeping tension off their bases. We are making our apposition very exact because that gives the best results in this work.

To fill up small cutaneous defects in the face we sometimes make a completely detached graft instead of a pedicled flap of the entire skin thickness. Nutrition of this detached flap then takes place by osmosis, exactly as it does in the case of Thiersch grafts which contain only epidermis. Similar free transplantation of the entire thickness of the skin is sometimes successful on the palmar surface of the hand, but much less frequently so on the dorsum.

Thiersch grafts for this hand would be much easier to make than these pedicled flaps, but, remember! a Thiersch graft gives a very poor pressure surface, and the hand must be able to stand hard knocks and usage. A Thiersch graft does not produce a surface that will stand as much subsequent flexion and trauma as the pedicled graft does.

These cases are treated with a dry dressing. All our grafts we keep entirely dry. I have the hand locked firmly in place with two strips of adhesive plaster, so that the flaps cannot be torn loose or put under tension.

We are dusting the surfaces over with bismuth subiodid. It has no antiseptic value at all, except the antiseptic action of the desiccation produced, which is always of some moment. That hand cannot spread any more. Finally, we put a wide strip of adhesive across the whole arm and around the body. That fastens our little patient's arm securely and keeps him from drawing his hand away from the flaps.

VISITING DOCTOR: How long will you leave that dressing on?

DR. MURPHY: Ten days. We just cover the hand all over with cotton, and let it stay dry. One can lift off the cotton dressing and look at the hand any time one desires. We treat all our pedicled grafts the same way—all with a dry dressing.

There is enough elasticity in these pedicled flaps so that they will spread and spread and compensate fully for any cicatricial contraction which may subsequently occur.

[NOTE.—The thumb flap partly sloughed, but not sufficiently to impair its utility, and full extension is easily possible. The index-finger flap lived in its entirety, and after it was detached and was entirely healed half of it was detached and thrown into the webbed space between the index-finger and thumb in order to insure continued freedom of motion in that angle. Primary union ensued and a good functional result.—ED.]

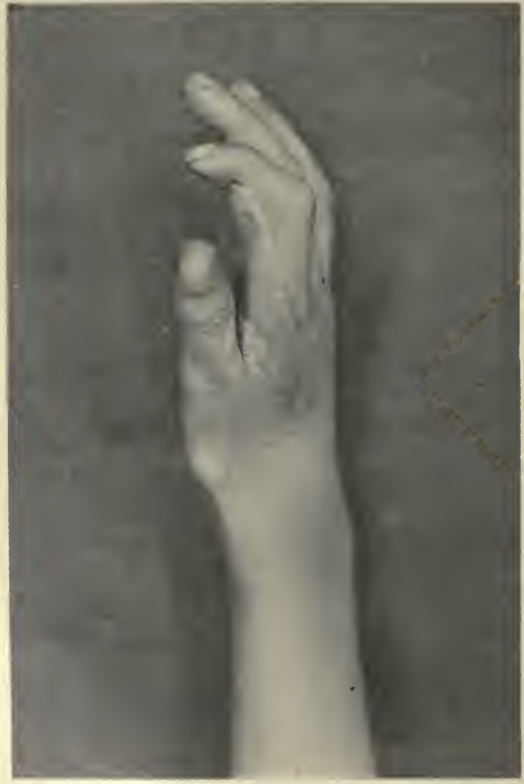


Fig. 33.—Contracting cicatrices of the index-finger and thumb. Photograph made shortly after the transplanted skin-flap was completely healed. Note the absence of any tension on the new skin.

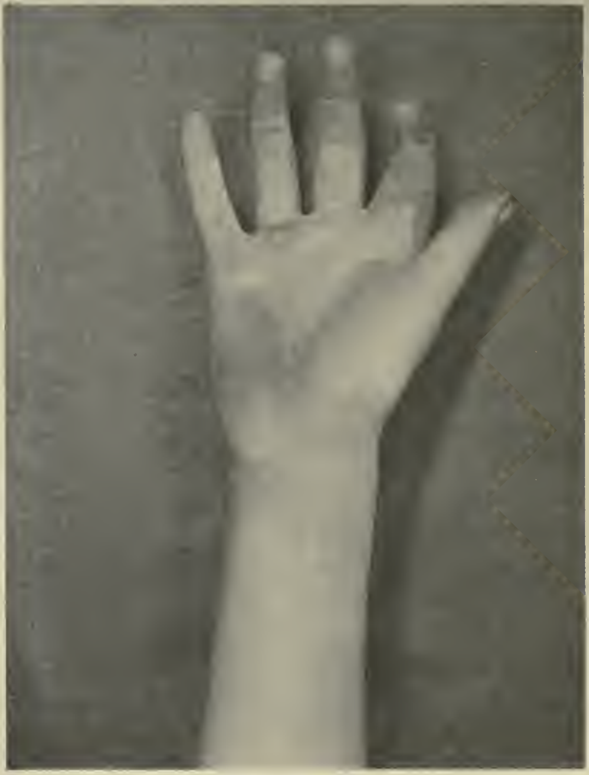


Fig. 34.—Contracting cicatrices of the index-finger and thumb. Photograph made shortly after the transplanted skin-flap was completely healed. Note the absence of any tension on the new skin.

LACERATED WOUND OF THUMB—EMERGENCY CASE

Dr. Murphy (September 5, 1914): This is an accident case, the result of catching the right thumb in a planing mill. The ulnar side of the patient's thumb has been torn away, and most of the bones of his thumb crushed and carried away. There still remain, however, a distal fragment near the tip of the thumb and a proximal bony stump. The greater part of the dorsal soft tissues is still remaining, and the blood-supply is intact, as shown by the oozing from all the torn flesh. The nail, too, has been torn off, but the greater part of the nail-bed remains. That will be a handicap to us rather than an advantage.

The injured hand has been soaked with iodin already, but I am not touching it with my gloved hand. I am using instruments entirely. What shall I do with it? This man is a laborer, and the value of his hand largely depends on the preservation of his thumb. Without a right thumb he is practically out of commission as a day laborer; but if he retains even the proximal joint of his thumb with the stump of the bony phalanx, he may remain an efficient workman. The more we can save for him, the greater the usefulness of the hand.

This injury has been handled in the usual way, the wound swabbed out with iodin without preliminary washing or scrubbing. Then the rest of the hand was swabbed with iodin and covered with a sterile roller bandage, so far as possible. He has already had his antitetanic serum, and is now ready for operation. What shall we do with this thumb to make the thumb, and consequently the hand, as useful as possible? The rest of his hand is in good shape. It is practically uninjured. I believe that the skin-flap which is most of what is left of the thumb will still live. What has become of the bone? It has been crushed as far back as the proximal articulation, and most of the fragments have been torn away with the flesh. The artery, the princeps pollicis, can be seen pulsating where it has been crushed off near the base of the stump. This obliteration of the artery by crushing explains why the patient has not lost more blood as the result of this severe injury. Crushing injuries often produce their own hemostasis in this way. To ligate the artery now is, I believe, not essential.

What shall we do with this stump? Any kind of a thumb is better than none. That is the immediate proposition. How much can we save? Practically all that is left, I believe. I shall place the tip of the flap in approximation with the base of the flap, letting the distal fragment of bone come in contact with the proximal bony stump, and suture the skin edges loosely together. See what that means! If I thus secure a thick thumb of flesh, I can fill in a bone transplant at any time. If we have no sloughing, and this wound heals by primary union, we shall have very much more skin-flap than bone. I can, therefore, later take a piece from his tibia and insert it in the fleshy stump to make the latter stiff. The nail-bed thus will have a new and strange position at the ulnar side of the base of the thumb. We shall have to take care of the nail-matrix later, because we do not care to have a nail develop in this useless and awkward position. These tag ends of flesh and bone into which dirt and grease have been ground must be clipped away because they threaten the integrity of the flap from their liability to infection and necrosis. If this flap will unite, as we believe it will, and as it does commonly in such hand injuries, we ought to secure a good result. Remember that the tissue here at the tip of the thumb preserves its vitality longer than any other tissue in the body except the tip of the nose. The tip of the nose lives still longer. That has been an old surgical observation. There is a story related in Dieffenbach's surgery—that of two men who were fighting, and one bit the nose off the other. It happened on the morning of Dieffenbach's clinic, and the injured man was brought into the clinic. Dieffenbach asked for the nose, and sent two students out to hunt for it. Just as they reached the spot where the *mêlée* occurred they saw a dog carrying off the nose. They chased the dog, got the nose away from him, and brought the nose back. Dieffenbach cleaned

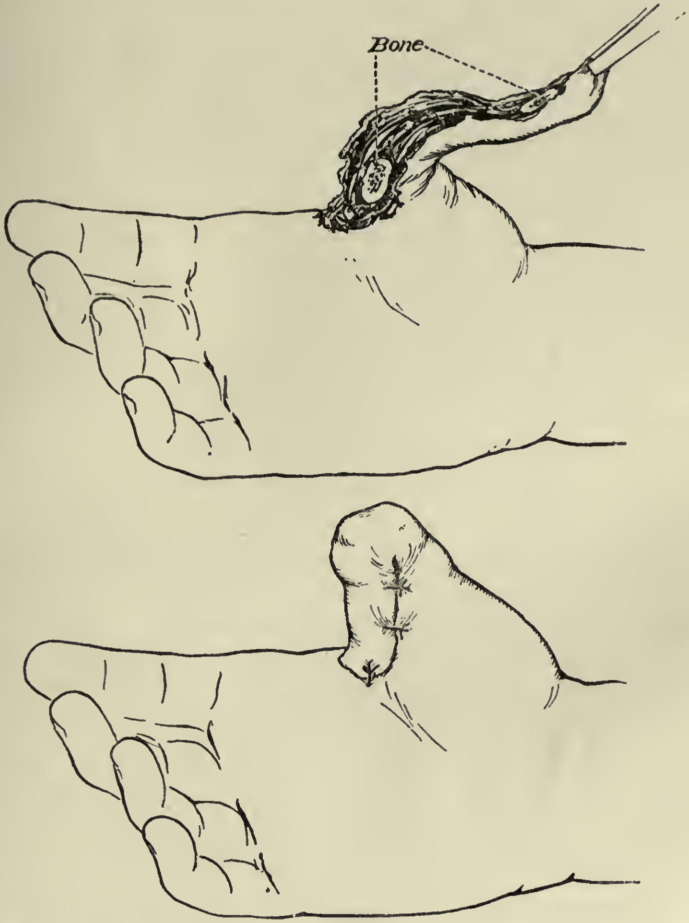


Fig. 35.—Plastic on the thumb following a tearing injury, a planing-mill accident. Note that the injury left a large part of the soft tissues of the thumb and their vascular supply, but tore away most of the bone. After cleaning the wound, cutting away the frayed and necrotic edges of the torn soft parts, and removing the loose bone splinters, the soft tissues were apposed with a few sutures and with only a strand of rubber tissue as drainage, thus leaving a large fleshy stump of the thumb into which later bone will be transplanted from his tibia to make a new phalanx. The very useful thumb thus produced will be invaluable to him in grasping objects, for he is a laboring-man and his livelihood depends on his being able to do heavy work.

it off, sewed it on the raw stump, and it grew. There is no doubt as to the fact that it *should* grow, because there are many other similar cases on record. When I was a student in Heidelberg one of the students had his nose cut off in a dueling match. The attending surgeon (Paukdoktor) was an old man, not noted for his cleanliness, and he sutured the nose back on. The boy was brought over to Czerny's clinic soon afterward, and Czerny took off the severed tip, freshened it, sewed it back on, and it grew. It might, perhaps, have united as well without this extra attention. The severed tips of the fingers grow back on in about the same way as the tip of the nose when surgically treated in the proper way.

Now that we have sutured these two bone fragments together we turn in this fleshy flap to cover up the gap, and suture it loosely in place with a few well-placed stitches. If the flap lives, the patient will have a serviceable thumb, which means a livelihood for him. For heavy work it increases his efficiency about 30 per cent. of what it would be without the preservation of this stump of the thumb. The one danger in these cases is the danger of infection, particularly in this location, where the tendon-sheaths of the thumb have been opened and the thenar fascial space exposed. We shall, therefore, insert a small gutta-percha drain, which will be removed in twenty-four hours if no infection occurs. We dress these cases with dry gauze. Wet boric dressings, by their moisture and heat, would only encourage the development of microörganisms and subsequent suppuration.

[Note.—The wound healed by complete primary union and the patient left the hospital in a week. The gutta-percha drain was removed at the end of twenty-four hours. The patient is so well satisfied with his present thumb that he has not yet returned for the insertion of a bone transplant.—Ed.]

MALUNION OF A FRACTURED FEMUR WITH GREAT ANGULAR DEFORMITY.—OPEN REDUCTION AND PLATING.—HEMORRHAGIC CYST AT THE FRACTURE SITE

HISTORY

THE patient, a boy aged nine years, a doctor's son, entered the hospital October 26, 1914. His present trouble dates back to an accident seven months ago—in March, 1913. The patient was swinging from tree to tree with a rope and pulley when the rope broke and he fell about 20 feet to the ground. He had immediately a severe pain in the right thigh and was unable to arise from the ground unassisted. Upon getting up it was found that his foot was everted and that his right thigh was about two inches shorter than the left. He was unable to stand on account of the pain. The fracture was not compounded. The patient was put to bed and a posterior splint was applied for one night. On the following day he was given an anesthetic and reduction was made. The limb was placed in a pneumatic splint with an extension attachment, and there it was kept for five weeks. At the end of five weeks the boy was up on crutches. Two months later the crutches were discarded, and it was then discovered that there was a bowing anteriorly at the middle third of the thigh.

The *x*-ray pictures taken recently show a malunion with an anteroposterior bowing of the femur.

COMMENTS AND OPERATION

DR. MURPHY (October 28, 1914): The *x*-ray plates of this case illustrate forcibly an important lesson. Of the two plates which we made this morning, the anteroposterior one shows an almost perfect alinement apparently, except for the bulging produced by the excessive callus at the site of fracture. The lateral picture, on the other hand, shows a marked degree of angular deformity—so marked, in fact, that because of it and the shortening of the limb the patient comes here for operative relief. Neither of these two *x*-ray plates shows much bone detail; but that is not the fault of the radiographer. It is owing to the fact that the two pictures were taken in the patient's own room in the hospital, using a small portable coil which necessitated so long an exposure of the plate that the little patient was unable to remain perfectly quiet during the entire procedure, and so blurred the details of the pictures by his slight restless movements.

The third plate, which was made in the patient's home town so soon after the injury that no callus formation had yet occurred, gives us no real idea of the extent of the deformity then existing, because it is only a one-way picture—anteroposterior; except for the overlapping and shortening, it seems to indicate a fairly good position of the fragments; but a lateral radiogram might have told us an entirely different story.

To attempt to judge the position of a fracture or the location of a foreign body, or to attempt, in general, to make a three dimension diagnosis by a two dimension plate, is to invite disaster.

There are certain locations in the body where it is so difficult to secure satisfactory two-way plates that the latter are impracticable. The hip-joint, the shoulder-joint, and the spinal column are examples in question.

In these locations one gains a better insight into the pathology present by stereoroentgenograms than by the two-way plates which have to be taken at not very radically varying angles. Stereoplates are useful in a great many other situations, particularly where the pathology is intricate in three dimensions; but in the locations mentioned above they are of great value to the surgeon, no matter how skilled he may be in reading single plates.

I have been insisting for a number of years that it is distinctly inadvisable, as a rule, for a doctor to assume complete management of the illnesses arising in his own family. The present is a case in point. This child suffered from too much leniency in sick-room discipline and from overattention to its comfort and desires.

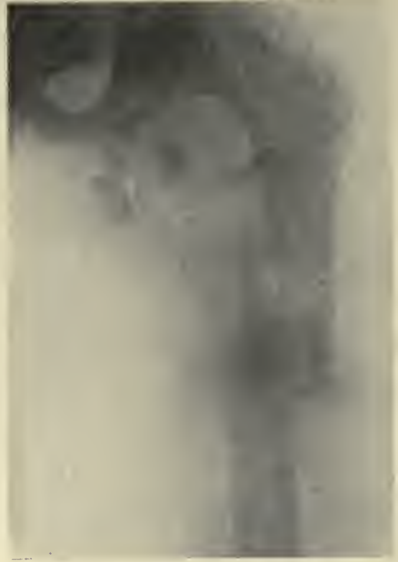


Fig. 36.—Fracture of the right femur. Anteroposterior roentgenogram made soon after the original injury, and before operation (April 15, 1914). Note the overlapping of the fragments and the considerable shortening of the femur produced thereby. The lack of bone detail in this picture is due to the fact that it was taken through the dressings and that a small portable coil was used, thus necessitating a long exposure during which the young patient was unable to remain perfectly quiet.

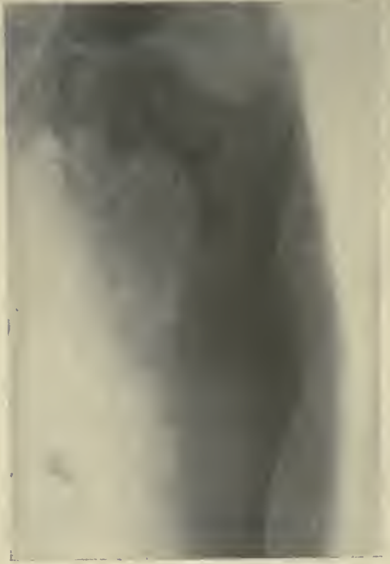


Fig. 37.—Old fracture of the right femur. Anteroposterior roentgenogram made October 28, 1914. Note that the marked angular deformity shown in the lateral view does not appear in this plate, which, accordingly, gives a deceptive appearance of the condition present. The alinement of the limb from this direction appears nearly perfect except for the bulging callus at the site of the fracture. The absence of bone detail is due to the fact that the picture was taken in the patient's room in the hospital by using a small portable coil; and the long exposure of the plate thus necessitated was for a longer period than the little patient was able to remain quiet.

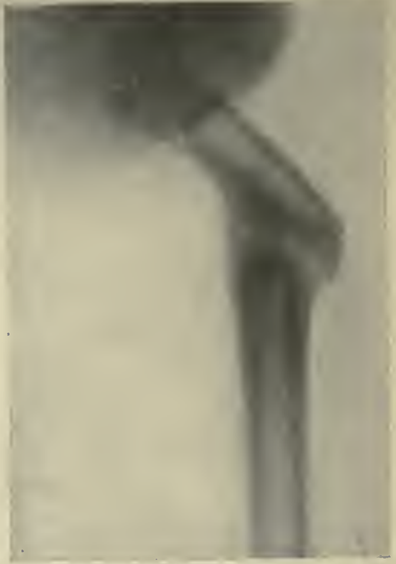


Fig. 38.—Old fracture of the right femur with marked angular deformity and anterior bowing. Lateral roentgenogram made on the day of operation (October 28, 1914). Contrast this plate with the anteroposterior picture. One would scarcely think them to be from the same patient. This case illustrates the necessity of two-way plates in all fractures and dislocations. This plate was also taken in the patient's room in the hospital with a small portable coil.

The patient should have been confined strictly to bed for four or five weeks, with longitudinal or upright extension, and then some splint immobilization. Instead, he was put early into an ambulatory splint because he was more contented so and less difficult to care for. An ambulatory splint is almost worthless for a child, because a child's incessant activity gets the apparatus continually out of adjustment, and the child either does not know when readjustment is necessary, or hesitates to speak of it for fear of being caused more pain. It is difficult enough to keep a child's fractured thigh straight when full extension is applied to it and he is flat on his back in bed, with none of his body weight resting on the limb. With the child up and at play, using the broken limb constantly and subjecting it to the risk of further trauma, a successful result is a remote possibility.

This was not a green-stick fracture of the femur—it was apparently a complete fracture. It is going to be rather a difficult case to handle, I am afraid, because of the degree of contracture which has already developed in the adductor muscles. The adductors pull like a bow-string on the bow which this angular deformity of the femur makes. They are still further contracted because of the overriding of the fragments which has taken place.

If we are to secure a successful result here, not only must the bow—the femur—be elongated, but also the string to the bow—the adductor muscles. When we have straightened the femur and applied the Lane plate, this strong adductor group will continue to pull vigorously against the plate, and will tend to bend it, but without effect, we hope. It means, also, a very considerable strain on the screws as well as on the plate.

This is not a suitable case for a medullary graft, because the latter can be very easily fractured while putting it in place under these circumstances. The patient has a good firm union, but in a bad position. If this were a case of non-union, we should put in a medullary graft; but it is not a non-union.

We are making a long incision in this case, and are placing it on the external surface of the thigh and slightly posterior, with the point of fracture at about its center. We are so placing it in order to go between the flexor and extensor groups of muscles in reaching the bone, instead of cutting through either of these muscle groups. Such a long incision is needed because it is going to be necessary to separate the muscles from the ends of the bone fragments over a considerable distance, in order to diminish the leverage which they will exert on the fragments after the latter have been reunited by the Lane plate. In addition to this precaution we shall use a strong plate and keep the limb under heavy traction, 15 or 20 pounds at least, until it is secure in the plaster-of-Paris cast. Where there has been as much callus thrown out around the point of fracture as there has been here, a good union usually follows the operation.

We know no way by which to insure union positively. All we can do is to place the fragments in accurate apposition and fasten them there. Apposition by itself seems to have but little control over osteogenesis, for quite often bone-ends which are in perfect apposition—for instance, with the bone-plate—show no attempt at union. But where a well-marked effort at union has been made by the fragments before operation, the rule is that union results after operation. The union may be a little retarded, but it ultimately occurs.

If the tension of the soft parts on the fragments is too great after they have been reduced, we shall apply a heavy cast to the limb and to the body as high up as the costal margin, holding the limb in a slightly everted and superlatively abducted position.

Judging by the bowing of the limb and by the *x*-ray findings, it seems likely that the intermediate callus was not fully and compactly formed in this case, although the present callus seems firm enough.

[Dr. Murphy made an external incision, extending from a little behind the trochanter half way down the thigh.]

See what an enormous incision one has to make in this case! There is the gluteus medius muscle at the upper angle of the cut. I am incising the fascia lata parallel to the long axis of the underlying muscle-fibers. Now I am approaching the bone by separating the muscles along their intervening planes instead of cutting through the fibers. This procedure gives a better functional result later than does the cutting of the fibers.

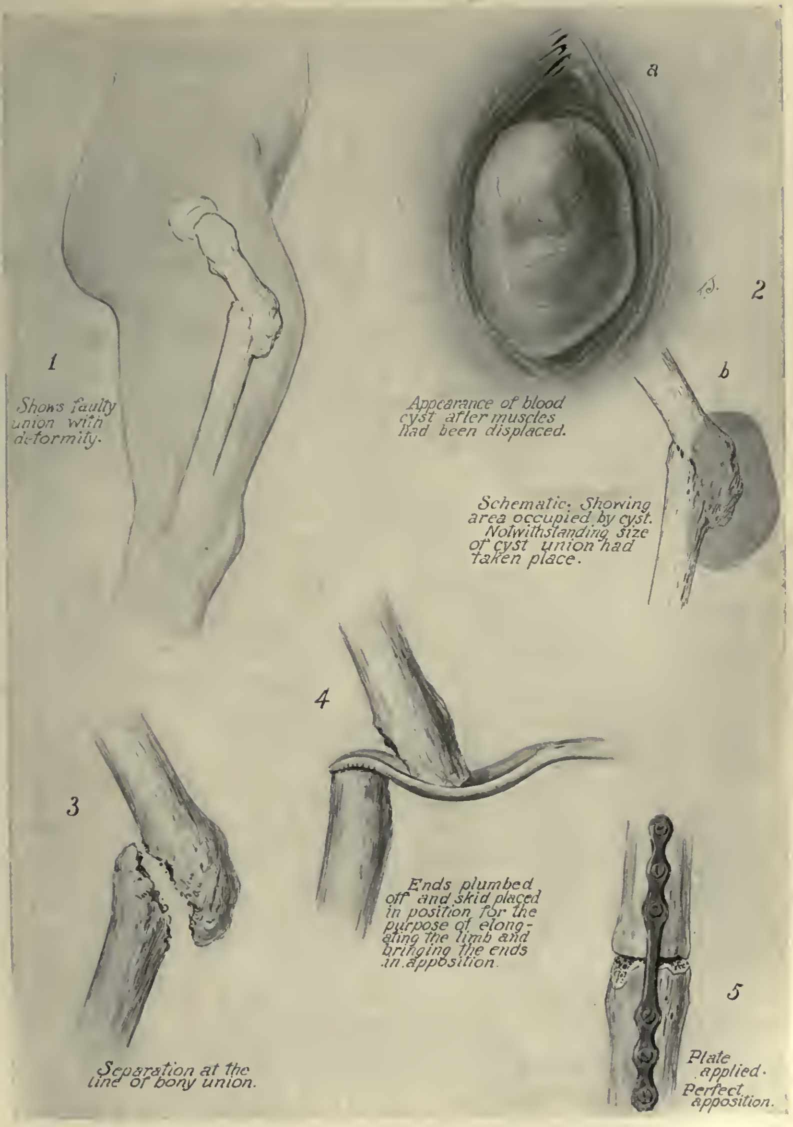

1
Shows faulty union with deformity.
a
2
b
Appearance of blood cyst after muscles had been displaced.
Schematic: Showing area occupied by cyst. Notwithstanding size of cyst union had taken place.
3
Separation at the line of bony union.
4
Ends plumbed off and skid placed in position for the purpose of elongating the limb and bringing the ends in apposition.
5
Plate applied. Perfect apposition.

Fig. 39.—Malunion of a fractured right femur with great angular deformity (anterior bowing). Open reduction and fixation with a Lane plate. Large cyst of hemorrhagic origin found at the fracture site.

Close to the bone here I have run against a curious pathologic structure. It is a hemorrhagic cyst. I am exploring its entire surface very carefully, looking for infiltrates or tumor nodules. I can find none at all. Therefore, it is probably not a malignant cyst, a beginning sarcoma, but the result of a large hemorrhage at the fracture site. The hemorrhage has developed into a cyst instead of being absorbed, probably on account of the excessive motion which was permitted to occur nearly at the site of fracture.

If you will read over the article which I wrote some years ago on the subject of non-union, you will note that I mentioned cyst formation as one of its causes. That this cyst, however, was the cause of the rather slow union here is highly improbable, I think, because at no point does it penetrate between the bones. It seems to have developed along the line of cleavage between the extensor and flexor groups of muscles, and is adherent to all the structures in the neighborhood.

The presence of this cyst somewhat complicates our operative procedures. It will be necessary, as well as desirable, to enucleate it, because it quite blocks our approach to the site of fracture from this direction.

I am splitting the cyst wide open in order to follow up its ramifications the better. It extends quite a distance around the bone to the inner side, where it reaches the level of Hunter's canal. Now that the cyst-wall has been fully exposed, the greater part of our task is over. I am paying particularly close attention to the dissection of the inner portion of the cyst in order to be certain that no fragments of it are allowed to remain. Any part that might be left would be very liable to continue to secrete, like any hygroma, and thus endanger union as well as asepsis. In making this dissection, I have been very careful not to traumatize the muscles surrounding the cyst.

This is the first cyst of this class that I have ever seen on the surface of a fracture. The others have always been in between the ends. It seems to have developed like a hygroma or a bursa from the friction of the muscles on the fascia and fat, the hematoma forming the original cavity, and the subsequent clot adding a foreign body irritation as well as a further source of friction.

This natural mechanism by which hygromas and bursæ are formed constitutes the mechanical basis on which we have developed our arthroplasty operations, the pedicled flap of fascia and fat producing a hygroma when exposed to friction, this hygroma varying in size with the size of the flap used and the direction of application of the friction. Such a hygroma soon becomes lined with endothelium and becomes a remarkably close histologic imitation of a joint, as well as a successful functional substitute for one.

In continuing this dissection and the exposure of the bone-ends, we are spreading the muscles apart, not cutting them. Now the site of the fracture is well exposed. Note how great is the angular deformity and with what readiness this sharp knuckle may be made to protrude through our incision. We are freeing the muscles from the bone-ends over a considerable distance. Note that we are also following up the vessels carefully and freeing them before we are making any attempt to straighten the bone. We wish to rid ourselves of all possible complications from the side of the soft parts before attacking the bone.

Our next task is to separate the bones. Here is some fibrous tissue in between them. It looks as if a strip of periosteum had intervened between the bone-ends, retarded union, and allowed this groove to form. I must grub out this mass of intervening fibrous tissue before I attempt to freshen the ends of the bones. No doubt it added its quota to the retardation of union.

We are separating the periosteum from the bones over quite a distance, so as to be able to have the Lane plate lie subperiosteally. Callus formation seems to be materially increased by the elevation of the periosteum from the bone for a considerable distance on both sides of the line of fracture.

The history states that there are two inches of shortening of the femur present, and I should judge, from appearances here, that that statement is not an exaggeration. We have refractured the bone as nearly as possible in the old line of cleavage, and we are freshening the ends before making extension on the limb and sliding them into contact with each other with our bone skid. All this work must be done without hand contacting, and that is

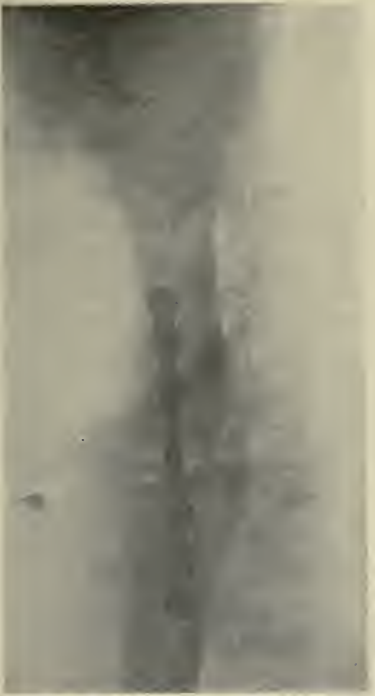


Fig. 40.—Old fracture of the right femur with marked angular deformity. Open reduction and application of a Lane plate. Anteroposterior roentgenogram taken through the plaster cast twenty-five days after the operation. Note the excellent position of the fragments and the well-advanced callus formation.

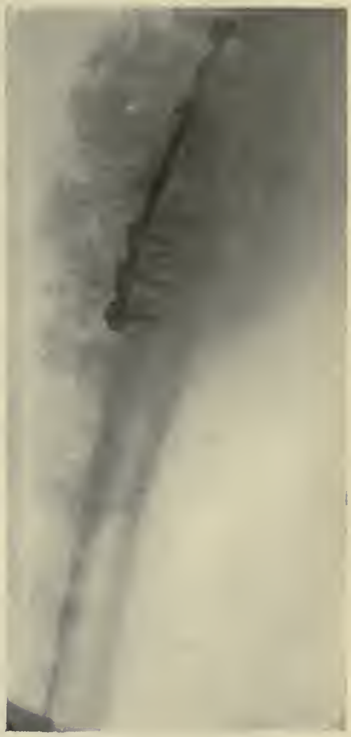


Fig. 41.—Old fracture of the right femur with marked angular deformity. Open reduction and application of a Lane plate. Lateral roentgenogram taken through the plaster cast twenty-five days after the operation. Note the excellent position of the fragments and the well-advanced callus formation.

what makes our technic at times appear rather slow and awkward. The temptation comes frequently in this line of work to put the gloved hand in the wound and hasten progress; but we have learned little by little to overcome this temptation, and prolong our operating time slightly for the sake of securing the perfect asepsis which absence of hand contact insures.

We are putting in a heavy, six-screw, vanadium-steel Lane plate. These screws are not easily adjusted, owing to the tendency of the soft parts to pull the bone-ends back into their old position. . . . At last the holes have all been drilled and the screws inserted.

We are drawing the stripped-up periosteum and muscles back around the bones and the plate. We are very carefully resuturing the fascia lata with a continuous catgut suture in order to avoid the formation of a muscle hernia through this aponeurosis. We are also uniting the skin with a catgut suture, so that we shall not have to disturb the dressing until we remove the cast.

It is just at this stage of the procedure that great care must be exercised not to bend or strain the plate. An assistant must devote his entire attention to making firm traction and abduction on the limb, and to seeing that no force is applied to it which would tend to throw it out of plumb. When the limb is once in the cast, it is safe; but during the period from the time when the plate disappears from sight as the wound is closed until the cast has hardened, the integrity of the plate and the correct alinement of the limb are in constant danger.

We are dressing the limb in superlative abduction with slight external rotation. The plaster cast which we are applying over the dressing will include the entire limb down to the toes, and also the trunk up to the costal margin, allowing room for a "dinner pad." The cast will be trimmed in the usual way to avoid friction with the skin at its edges. Note that we are cutting out a window on the external surface of the leg just below the knee, so as to avoid pressure on the external popliteal nerve. This is a precaution which we never omit in applying a leg cast. It is part of our regular system. The penalty of neglecting it is the development of an occasional case of foot-drop, for which the patient is certain to blame the surgeon, as he is thoroughly justified in doing.

Note that we carried out our entire operative procedure without any gloved hand or the touched portion of any instrument, sponge, or ligature contacting with the wound.

[Pathologist's Note (Dr. W. O. Sweek).—The wall of the excised cyst shows organizing fibrin and round-cell infiltration, but no evidence of malignancy, and resembles somewhat a vessel-wall in cross-section, because of the presence of a lining endothelium.]

[Note.—The wound healed by complete primary union. The bone reunited promptly with abundant callus and good physical conformation. The patient left the hospital at the end of six weeks still in the leg and body cast, which is to be removed at the end of eight weeks. *One of the most common errors in the management of fractures of the femur is the too early removal of extension or mechanical supports.*—Ed.]

A TALK ON A CASE OF GANGRENOUS APPENDICITIS OPERATED THE PREVIOUS EVENING

Dr. Murphy (September 10, 1914): I want to talk to you for a moment between cases about the patient to whom this gangrenous appendix belonged. [Shows specimen.] What was her temperature when we saw her yesterday, Dr. Hochrein?

Dr. Hochrein: Her temperature was normal.

Dr. Murphy: This patient came into our office late yesterday afternoon. She gave a somewhat indefinite history, some points of which were quite suggestive of an acute attack of appendicitis. She lives out south of the city, and came in on the train yesterday unassisted. She complained that she had had a mild attack of abdominal pain last Sunday (September 6)—nearly all day. It was not sharply localized, and was not at all severe. She had a normal temperature at the time, she thinks, but she did not measure it with a thermometer. She had a little nausea on Sunday night.

Since Sunday the pain in the abdomen has continued to be annoying, especially on the right side, and it was because of this pain that she came to consult us. We saw her about 6, or possibly 5.30 yesterday evening (Wednesday). On examining her abdomen we found a rather distinct sensitiveness over the appendiceal area, but she had a normal temperature. We then had a leukocyte count made, which revealed 14,500 leukocytes to the cubic millimeter. The chief reason that I have for citing this case is to call to your attention the fact that in cases of appendicitis where there is an absence of fever the leukocyte count may be of great help in making the differential diagnosis.

I urged the patient to go directly to the hospital, and while in the office she agreed to do so. After she left the office, however, she decided to go home and talk to her sister. She walked from the office to the Twenty-sixth Street station of the Illinois Central, a distance of five blocks, and, while waiting there, she had a little additional pain; then she fainted and fell on the station floor. When she came back to her senses she told the station agent that she had come from our office. He telephoned to us and we sent the ambulance over to the station and brought the patient to the hospital, where we took out her appendix the same evening.

[Demonstrates the specimen to the clinic.]

This specimen does not present so striking an appearance now as it did last night when we had just removed it. This is an acute appendicitis of the gangrenous type. When we saw it last night the appendix was much distended, and this dark area here was almost ready to perforate into the free peritoneal cavity, because, although the tip was already adherent, the proximal and gangrenous portion was not adherent to anything, and the pus, some of which you still can see inside the lumen, would have been discharged into the free peritoneal cavity in the course of a very few hours more. Note well this dark gangrenous zone, the place where the appendix was about to perforate. And yet the patient had a normal temperature and a normal pulse! *But she also had a leukocytosis!* That is the important factor which I wish again to emphasize. If the entire appendix had been gangrenous, she would not have had so high a leukocyte count as 14,500 yesterday, nor so much pain. It was the pus in the distal and living portion of the appendix which, by its absorption, produced the leukocytosis.

The lesson we should learn from this case is that while the early symptoms in appendicitis indicate clearly the nature of the trouble to begin with, the later symptoms often fail to indicate the malignant course which the pathologic process is pursuing. The four cardinal symptoms of pain, nausea and vomiting, elevation of temperature, and local sensitiveness appear early and usually in the order named.

This message is timely and, as a rule, unmistakable. It says, "*Operate, and operate now!*" But woe to the patient if he or his doctor fails to heed this first warning! The pain, the vomiting, and the fever may next appear only after perforation has occurred, when they herald a spreading peritonitis and impending death.

The patient stated yesterday that she had felt feverish the day before, but the fever had subsided before she reached the office, and she was feeling so much better *because her appendix was already gangrenous and, therefore, insensitive*, that she declined our advice to enter the hospital at once; and if we had not had her leukocyte count made, we ourselves might have been a little lukewarm about insisting on her staying at once at the hospital last night to have the immediate operation. Fortunately for her, she soon was brought back to the hospital to furnish us this striking object-lesson, which one must see to appreciate.

The patient is having a little distress this morning with gas pains; but she has no fever, and is very happy at her narrow escape, and that she has her operation to look back on rather than forward to.

[NOTE.—The patient made an uninterrupted recovery.—ED.]

[NOTE.—In connection with another case of gangrenous appendicitis on p. 1101 of the December, 1914, CLINICS, the last sentence on the page states, "If you open the abdomen and find an abscess, be happy to let out the pus, close the abdomen, and let the appendix remain." For clearness' sake it evidently should have read, "Close the abdomen down to the drain," for we have already had an inquiry regarding this statement. Dr. Murphy always drains an appendiceal abscess.—ED.]

14b

RICHMOND STEPHENS, M. D.

THE CLINICS
OF
Volume IV JOHN B. MURPHY Number 2

MURPHY'S CLINICAL TALKS ON SURGICAL AND GENERAL DIAGNOSIS

A DIAGNOSTIC TALK ON OSTEOMYELITIS

DR. MURPHY: While our next operative case is being prepared, Dr. Kreuscher is going to use this amphitheater for an intravenous injection of neosalvarsan, and then will renew a nitrogen pneumothorax on one of Dr. Mix's pulmonary tuberculosis cases. Dr. Kreuscher has been doing all this line of work for us for some time, and we consider him very expert in it, as well as very careful. Those present who are interested in these procedures will see them better if they will come down to the front of the amphitheater.

For the benefit of the other visitors who are more interested in clinical surgery, I wish to show and discuss, while Dr. Kreuscher is working, two cases that are instances of the evil results which often follow a failure to diagnose promptly, and, therefore, to operate early, a common surgical condition—acute suppurative osteomyelitis.

The error is all the more lamentable in the first case, and the doctor's sin of omission is all the more aggravated because this little patient's own father was his physician and was with him and treated him from the very beginning of his illness. Right here I might drive another nail home by reiterating one of my favorite theses—that a doctor should not treat illness in his own family. In sickness in his own home the average doctor is not prompt enough or thorough enough in his diagnosis and therapy. He is tardy in recognizing the seriousness of a case, slow to make a thorough physical diagnosis, dilatory in carrying out the routine laboratory tests, and lax in his sick-room discipline. He is too much inclined to let the physical ailments of his family drift along and right themselves—if they will—just as he lets his own. The average doctor becomes so used to seeing physical disaster striking people all about him and leaving him unscathed that he unconsciously comes rather to believe himself immune to serious physical trouble, and carries over this feeling of immunity to corporeal ailments to his family as well. This careless over-confidence sometimes leads to sad results.

The history of this case is as follows:

The patient, a boy aged fifteen years, entered the hospital September 6, 1914. He has had the common diseases of childhood, and during 1911 had several attacks of malaria.

His present trouble dates back to January 5, 1914. During the evening of that day the patient felt a pain about one inch below the knee-cap, on the inner side of the left tibia. He does not recall having a chill at the time. There is no definite history of any preceding injury to the leg excepting that two weeks before the onset of the pain he fell from a tree, but did not hurt himself in falling or strike on that leg. He had had no cold in the head or other illness for several weeks previously.

The onset of the pain was sudden. The pain was not sharp in character, but it was sufficiently severe that the patient was awakened by it at close intervals during the ensuing night. The next day he walked to school, a distance of one mile. In the forenoon the pain below the knee became so annoying that the patient returned home before the close of the school session. The patient's temperature then was 102° F., but he had no chill during the day and no nausea. The upper third of the left leg was swollen and painful to the touch, and the swelling extended even somewhat above the knee-cap.

January 9, 1914, three days later, a new painful area developed over the upper right humerus, two inches below the shoulder-joint (that is, in the region of the metaphysis). Four hours later the shoulder-joint itself became swollen and the patient's temperature went up to 102° F. The pain became intense. It was treated with salicylates internally and hot-water bags externally. His condition continued about the same, with gradual improvement, until January 14, 1914, when he had another similar focus develop in the lower third of the left thigh. The pain here likewise was very acute, came on without a chill, and was soon followed by a temperature of 102° F. The left knee was not moved during the attack, as the pain thus produced was great; but it could be flexed voluntarily when desired; that is, the knee-joint itself was not involved in the process. During the third week following the onset of the affection the patient felt better, and on February 27 he was taken by his father to Hot Springs, Ark., where he took a series of baths, without being materially benefited.

On March 7, 1914, he underwent an operation. An incision five inches long was made over the lower third of the left thigh down to the bone; about two ounces of pus were removed and the bone was cureted. It was found that some destruction of the bone had occurred. Drainage openings were made on both the inner and outer sides of the thigh, and through-and-through drainage established by drawing a rubber drainage-tube through both the openings. His temperature was normal the next morning, but reached 100° F. again in the afternoon.

The central incision and the inner drainage opening are closed now. The outer opening still drains pus. A culture from the discharge shows *Staphylococcus pyogenes albus.*

On March 26, 1914, an incision was made over the anterior upper third of the right humerus, two inches below the shoulder-joint, between the deltoid and biceps muscles to the bone. The humerus was found rather soft for a distance of an inch or so. It was not cureted. A rubber drainage-tube was inserted, but was withdrawn that same night because of the pain it caused. The patient ran a low temperature for some time.

On May 16, 1914, he had a chill in the afternoon, followed by a temperature of 102° F. and a recurrence of pain below the right shoulder. The right humerus was operated on again and the bone cureted. After this second operation the patient's condition improved somewhat.

On May 25, pain developed again over the upper third of the left tibia. The patient had no chill, but a swelling soon made itself evident over the upper tibia. This swelling subsided, however, in twenty-four hours. On June 26 he had another attack of tibial pain which was very severe. Then a swelling again appeared over the upper third of the tibia. This swelling was opened. Some pus discharged, and the bone was cureted. A drainage-tube was inserted. After its removal a few days later pus continued to discharge from the drainage opening.

In going over this boy's case we were unable to locate the site of the primary infection. There is no history of a furunculosis, no history of an infection of his fingers or toes, no tonsillitis or pharyngitis, nor anything from which one can make out a starting-point for the metastatic lesions in his bones. His first complaint was the pain over the tibia just below the left knee, and within a short time—in fact, within twenty-four hours—he was completely incapacitated by his illness and was in bed, with a temperature as high as 102° F. He did not have a chill, either preceding or following the onset of the pain. The tenderness below the knee was marked on superficial pressure and immediately so, a fact which would rather indicate that the subperiosteal zone of the tibia was also involved at the onset, and not the medulla alone.

Let us consider how such a condition is brought about, and then we shall see why osteomyelitis runs the course it does, and how the symptoms produced depend on the pathology present in the individual case and enable us, with a high degree of accuracy, to recognize the pathologic changes present before cutting down on and exposing the bone.

Acute suppurative osteomyelitis is, so far as we know, always the result of a bacterial infection. The "spontaneous" variety, with which we are concerning ourselves at present, is probably always a metastatic infection from some recognized or unrecognized primary focus elsewhere in the body. This primary focal infection may be either acute or chronic. Its essential feature is that it allows pyogenic organisms to enter the blood-stream, where they circulate until they find a nidus suitable for their growth, for instance, a bone which has recently been subjected to a trauma and has a small hematoma or two or an accumulation of serum.

How do we know that these infections are caused by the pyogenic bacteria? And why do the microörganisms select the bone-marrow for their habitat?

We know that the pyogenic bacteria, especially cocci, are the causative organisms, because they have been repeatedly grown in culture from the pus of these cases; and these cultures being reinjected into animals, generally young rabbits, have reproduced the disease.

The organism most frequently found is the *Staphylococcus pyogenes aureus*—less frequently the *albus*. *Streptococcus pyogenes* also is found, besides *B. coli communis*, *B. typhosus*, and the pneumococcus. Other organisms than these are rarely found in osteomyelitic foci. These bacteria may be found in pure culture or as a mixed infection. The typical case of acute osteomyelitis is that which is caused by the *Staphylococcus pyogenes aureus;* and in speaking of acute osteomyelitis, its symptomatology and complications, it is the *Staphylococcus aureus* infection which we usually have in mind. This is the variety which runs the most severe local and general course, as a rule, and which, when not diagnosed early and provided with prompt drainage, results in massive bone necroses and pyemia.

The streptococcus infections are less liable to produce extensive medullary changes. These foci are more often cortical or periosteal; that is, the streptococci seem to prefer the denser areas of bone for their growth, and, accordingly, often locate in the epiphyses or near the epiphyseal line, so that joint effusions and epiphyseal separations are more frequent with streptococcus osteomyelitis than with the other varieties. Although the severe medullary phlegmon so characteristic of staphylococcus osteomyelitis is rarely seen in streptococcus infections, still a local necrosis preceded by an intense inflammatory edema is not rare.

Osteomyelitis occurring late in typhoid fever or during convalescence from typhoid is a mild variety, as a rule. In these foci typhoid organisms can usually be demonstrated in pure culture. Occasionally other organisms are also found, and sometimes the cultures are sterile. This variety of infection is a rather superficial one, the focus or foci lying in the cortex or subperiosteally. Operation is usually unnecessary unless the local

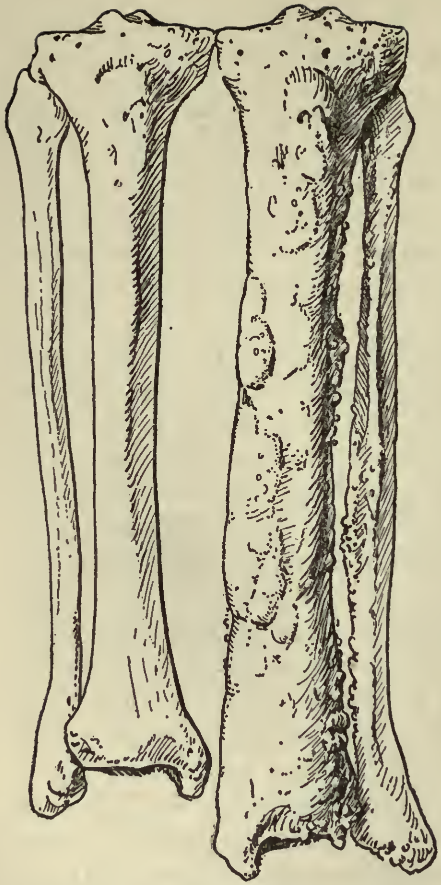


Fig. 42.—A typical untreated case of acute osteomyelitis of the tibia with necrosis of nearly the entire shaft of the bone and formation of an enormous involucrum. The pus finally found its way to the outside through several fistulæ. Note the increase, not only in width, but also in length, of the affected tibia. Note that in this increase in length the fibula has kept pace with the tibia. This increase in length is produced by osteomyelitis only in growing bones, where the medullary phlegmon reaches one or both epiphyseal lines and irritates or stimulates, but does not destroy, them. Possibly an increase in vascularity is also produced at the epiphyseal line by the process. The causative organism in this individual case is not known; but the organism most commonly found in such cases is the *Staphylococcus pyogenes aureus*. The fibula shows periosteal involvement (osteophytes), but no osteomyelitis focus. The corresponding bones of the opposite limb are included in the drawing for the sake of comparison. (Specimen from Dr. Murphy's bone collection.)

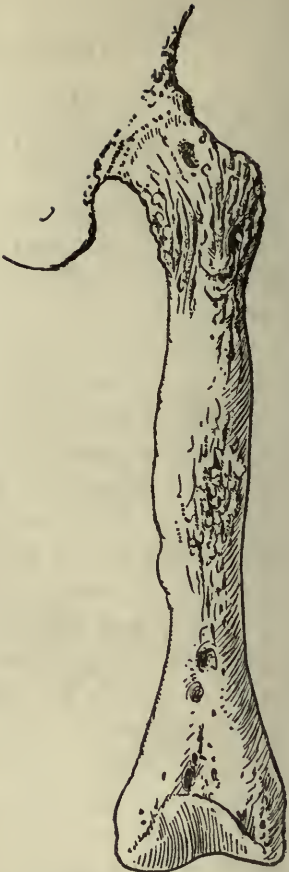


Fig. 43.—An old untreated case of acute osteomyelitis of the femur. In this case the pus broke through into the hip-joint and produced a complete bony ankylosis. Note the periosteal osteophytes and bone fistulæ in the neighborhood of the great trochanter. There is a very noticeable thickening at the center of the shaft, due to periosteal apposition of new bone, with the formation of periosteal osteophytes. The organism causing the infection is unknown, but, very likely, was the streptococcus, because of the severe articular affection in comparison with the rather mild osteal involvement. (A specimen from Dr. Murphy's bone collection.)

and general symptoms are very severe. The diagnosis of typhoid osteitis and periostitis is readily made by the clinical examination, and subsequently confirmed by the *x*-ray plate. The clinical manifestations run about as follows:

The convalescent starts to complain of pain over one or both shins, and, perhaps, also over one or more other long bones. Tenderness is elicited on examination over more or less of the entire length of the shaft. It is usually not very severe, and the patient's general condition is good, fever low or absent, and leukocytosis moderate or lacking. The first *x*-ray plate often gives a negative finding; but by the time the patient's complaints have lasted from two to three weeks the typical shadow of an ossifying periostitis appears and clears up the diagnosis. Sequestrum formation is distinctly rare.

In the days when I was a student in Rush Medical College we used to hear a good deal about "spontaneous" osteomyelitis and "cryptogenic" osteomyelitis. Among etiologic factors, "dirt," "unrecognized pricks and scratches," "passage of noxæ through the healthy skin," "winter months and infrequent bathing," used to be prominently mentioned. The really obvious fact that all "spontaneous" infections of the medullary cavity *must* arrive by the blood-stream was rather generally overlooked. It was the general recognition of the genesis of metastatic arthritis which started surgeons looking for focal infections as the metastatic sources of osteomyelitis also; and once that we started to hunt for them, we found them with ridiculous ease. I think that many of us had probably had the idea for a long time in the back of our heads that osteomyelitis *must* be always hematogenic in origin; but most of us never formulated the idea in definite terms until the metastatic nature of the multiple arthritides was well established.

It is a noteworthy and rather remarkable fact that focal infections are most apt to produce bone metastases, just as they produce joint metastases, when they have reached an afebrile or subfebrile stage. I have mentioned that typhoid osteitis and periostitis have a predilection for occurring during the early period of convalescence or during the last week or two of fever, when the latter is running relatively low. Following a furuncle or a panaritium, osteomyelitis develops usually when the original infection is almost, but not quite completely, healed. The streptococcus and pneumococcus infections come on just as the patient is getting up and about after his illness, or when he is already back at work after his bronchitis, acute pharyngitis, or tonsillitis.

Another feature worth mentioning is the fact that the patient's temperature in these cases does not start to rise until the bone inflammation actually sets in. There is no prodromal fever. So long as the organisms are circulating free in the blood and have not yet located a site for their propagation, the patient remains afebrile, as a rule, although we know practically for certain that he is suffering from a septicemia, mild though it be.

It usually takes a trauma or an injury of some sort to localize the general infection in a bone. This observation is well established, not only clinically, but also by experimental observation. Rosenbach produced osteomyelitis in young rabbits by injecting pyogenic cocci into their blood-stream and then producing subcutaneous injuries to their long bones. Ullmann accomplished the same result by ligating an extremity in infected young animals. Lexer and others have produced osteomyelitis in rabbits with staphylococci injections alone, without producing artificially any local change in the bone; but most animals without an artificial *locus minoris resistentiæ* so injected have died rather promptly of pyemia and metastatic suppurative nephritis before osteomyelitis pus foci have had time to develop. Pneumococcus osteomyelitis has proved practically impossible to produce in rabbits by the same means used to produce staphylococcus osteomyelitis. Nearly all the injected rabbits die promptly from the acute pneumococcus septicemia before osteomyelitic foci develop.

Why do these metastatic suppurations show such a predilection for the bone-marrow, especially in the region of the metaphyses and epiphyses? One reason is because the arteries in these locations are end-arteries. The septic embolus produces an area of necrosis, and in the dead area the microörganisms find a favorable location for their further growth. Furthermore, the vascularity of these regions in growing bone tends to suffuse these infarcts with blood and make them a still better culture-medium than adult bone. At any rate the clinical fact is well established that those metaphyses and epiphyses at which growth is most rapid and the blood-supply most abundant are the most frequent sites of osteomyelitis.

Now that we have gone somewhat into the etiology and pathology of acute osteomyelitis, let us correlate these facts with our clinical observations.

The osteomyelitis patient usually gives a history of an acute or chronic focal infection of some sort, or one finds evidence of it on a careful physical examination. Although from this focus microörganisms have begun to circulate in the blood-stream, they do not make their presence clinically manifest, very frequently, until some of them lodge as a septic embolus in the end of a long bone. This localization of the infection is usually associated with a trauma and often preceded by an exposure to the weather. The boy has been swimming or skating; he stretches out on the ground and rests there for an hour, exposed to a cold wind, or lying, perhaps, in the snow. He may not realize at the time that he has become thoroughly chilled. Or the boy may fall into the creek in the springtime, when he is riding a log in the ice water; and he walks home across the fields with chattering teeth. There is usually a localizing trauma of some kind, also, associated with the preceding infection of the upper air-passages. The night following the trauma the patient has a chill. The next morning he has considerable fever and feels unable to get up and go to work. In twenty-four hours after the onset of his trouble he is completely out of commission because the infective microörganisms arrested within a solid bony wall are producing their destructive metabolic products under pressure, thus making a severe local as well as a general reaction.

The inflammation lying deep in the bone, one cannot demonstrate its presence by superficial pressure. At least there is no tenderness on superficial pressure at first. It is only when the soft parts become invaded by the infection that superficial tenderness appears. Because the inflammation causes pain in the neighborhood of a joint it is diagnosed "rheumatism" by the general practitioner about nineteen times out of twenty. And yet the differentiation between the two conditions is relatively easy. There is no effusion into the joint early in an osteomyelitis. There may be an effusion present in the joint *late* in an osteomyelitis, when the focus of suppuration has ruptured into the articular cavity, as is frequently the case in streptococcus infections; but there is no fluid in the joint *early*—in the first twenty-four, forty-eight, or seventy-two hours of the infection, when correct diagnosis is so important from the standpoint of treatment. Furthermore, in an acute arthritis there is superficial tenderness over the joint, whereas in acute osteomyelitis the focus of tenderness lies over the bone, either above or below the joint, and this tenderness is only to deep and prolonged pressure—not to superficial pressure. Acute osteomyelitis and acute metastatic arthritis are rather alike in their early clinical symptoms, but they are readily differentiated by the physical findings.

As the medullary phlegmon, which is an acute osteomyelitis, spreads, it extends not only longitudinally inside the bone, but also rapidly works its way to the surface through the pulp-canals, and soon forms a subperiosteal accumulation of inflammatory products. The subperiosteal inflammation may then rapidly spread and separate the periosteum from the bone over a wide area. The bone, thus deprived of its internal blood-supply by the pressure of the marrow phlegmon, and likewise robbed of its periosteal vascular connections by the spreading periostitis dissecans, rapidly undergoes necrosis over a considerable extent of its shaft. The amount of necrosis depends on the virulence of the infection and on the promptness and thoroughness with which surgical measures are instituted.

This boy had a focus of necrosis in the shaft of his tibia, then another in his femur, and another in his humerus. The last-mentioned bone became necrotic for a distance of eight inches, and a large sequestrum was formed in the shaft.

We learned this morning that in traumatic myositis almost all the destructive mischief is accomplished inside the first forty-

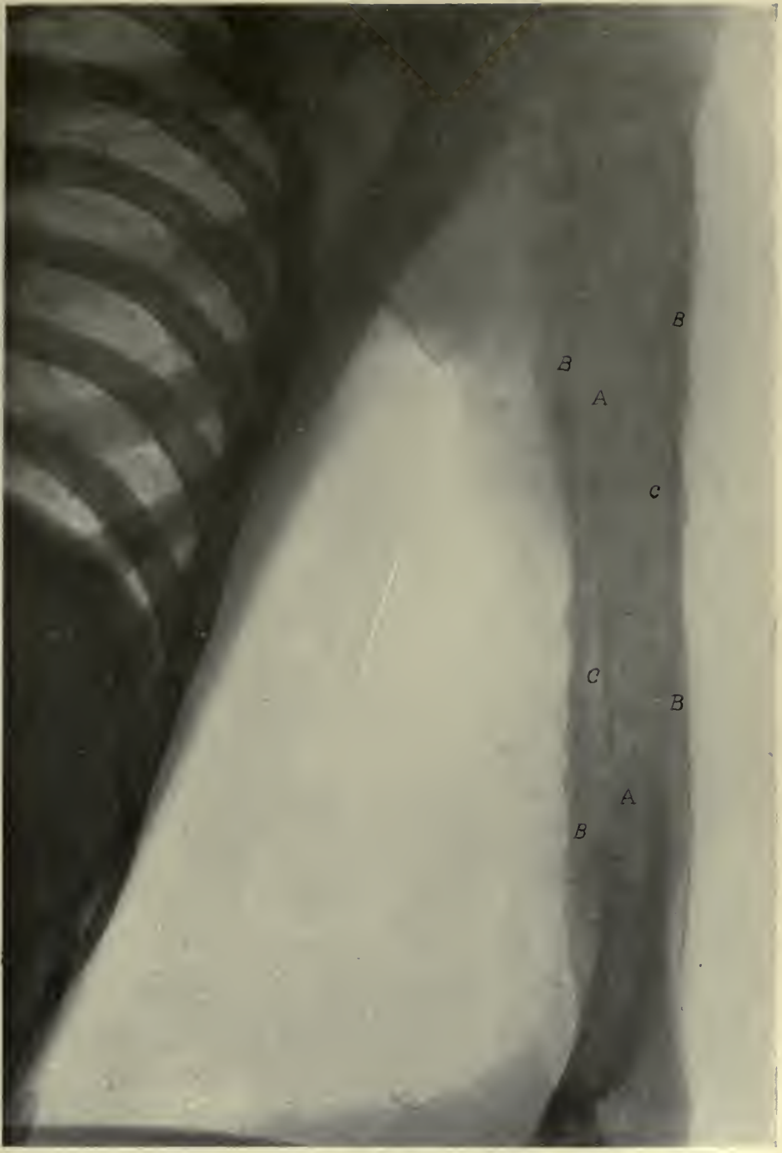


Fig. 44.—Acute suppurative osteomyelitis involving several bones, the result of failure to recognize promptly the primary osteomyelitis focus. Anteroposterior roentgenogram of the right humerus. Note the large sequestrum (*A*) involving over half the length of the original shaft. Note the great size and thickness of the involucrum (*B*) which has formed around the sequestrum. This involucrum will easily support the arm when the dead shaft is removed. Note the light zone of granulation tissue (*C*) separating the dead from the living bone. This sequestrum is now ready to remove and will require for its removal an incision in the soft parts and involucrum eight or nine inches in length. Note that the medullary phlegmon extended upward to the upper epiphysis of the humerus, but did not pass the epiphyseal line or invade the shoulder-joint. This is the rule with staphylococcus infections. It is chiefly the streptococcus phlegmons which occasionally pass the epiphyseal line and produce epiphysitis, epiphyseal separation, acute arthritis, and, still more rarely, spontaneous luxation of the joint.

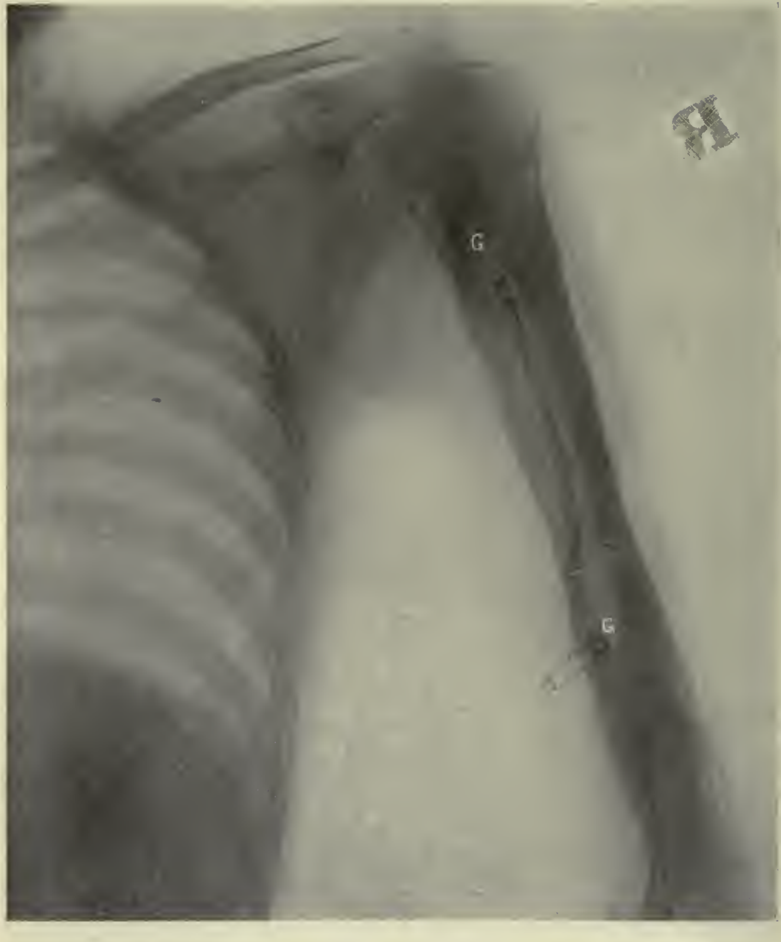


Fig. 45.—Multiple osteomyelitis the result of failure to recognize promptly the primary focus of bone infection. Anteroposterior roentgenogram of right humerus made soon after the operation. Note the long groove (*G G*) which had to be chiseled out of shaft in order to remove the sequestrum. The fenestrated rubber drainage-tube lies in the cavity formerly occupied by the sequestrum.

eight hours of the disease. If the surgeon is to be of service to the patient, he must relieve the intramuscular pressure inside that period of time; for otherwise the muscles will soon be destroyed by the pressure of the accumulated blood and serum. There is still greater urgency for the surgeon to act with equal alertness and equally radical positiveness in acute osteomyelitis if he is to be anything other than an expensive ornament to that patient's environment. The destructive pressure inside the bone must be relieved by the removal of the tension; that is, the marrow cavity must be opened at the site of the inflammation. It makes but little difference *how* it is opened, so long as it is opened promptly and the products of inflammation allowed to escape. The surgical situation is like that in a boil or in a phlegmon—take off the tension and the destruction stops.

It is not necessary to be elaborate in one's surgical technic in such cases. If general anesthesia is not available, it can be dispensed with. Local anesthesia is always available to the physician with a hypodermic syringe by injecting the skin, fascia, and periosteum with either normal salt solution, water, or even air under pressure (danger of embolism in vascular regions!), thus producing an infiltration anesthesia of only short duration, it is true, but long enough for our purpose. Then, with a gimlet, a small chisel, or even a screw-driver, work a passage through the cortex of the bone and let the pus out of the marrow cavity.

Because this boy did not have this relatively simple operation performed he suffered a necrosis of nearly half the shaft of his humerus, and also developed necrotic foci in his femur and tibia. Because of his father's failure to recognize and to treat properly this condition, the boy has lain at death's door several times since January. A little more use of cortical cells on his father's part would have saved him all this trouble.

If such a case comes to you *after* the early period of the disease, that is, on the third, fourth, or fifth day, you may not be able to prevent necrosis, but you may still be able to limit its extent and diminish the amount of subsequent soft tissue destruction and prevent the burrowing of the pus into the adjacent joint, the sloughing off of an epiphysis, and the development of a pyemia. All these disasters are well worth forestalling, and they can be forestalled by the simple surgical measures mentioned above.

If you do not receive the case until the end of the first week, however, when the bone is already destroyed and a large quantity of pus is present, then what shall you do? Just let out the pus and be satisfied. Do not try to remove the dead bone, for it is not yet separated from the living. Remember that the separation of the dead from the living bone is a function of the living bone, and is a process which requires time. The surgeon must wait until nature has separated the sequestrum from the living bone before attempting to remove the necrotic fragment.

Especially is meddlesome surgery to be avoided if the whole shaft of the bone is destroyed and the periosteum elevated, but not destroyed. Drain the pus, but leave the bone. Then, when the dead bone has separated as a sequestrum from its attachments, in four, five, or six weeks—*still leave it alone!* If such an extensive sequestrum were removed, nothing would be left to support the living periosteum which is still osteogenetic even when it is elevated from the bone in that way. If the sequestrum be removed, the periosteum will collapse and its two inner layers come in contact. Then very little, if any, new bone will be formed—certainly not enough to support the limb.

If the entire shaft is destroyed, permit that sequestrum to remain for months—six months, ten months, twelve months—until a complete involucrum of periosteal bone is formed entirely surrounding the sequestrum. Then keep on waiting until that involucrum becomes strong enough to support the limb; otherwise the latter will contract and deform from the contraction of the neighboring muscles which have lost their support.

When the time comes, however, to remove the sequestrum, then *remove it!* Do not cut down on the bone and make a hole scarcely large enough for a swallow to peep into. Make an incision long enough and wide enough to expose the entire sequestrum in all its ramifications. Expose it and remove it *in toto. Do not leave any fragments!* Any oversights, like allowing a small portion of the sequestrum to remain, means that a sinus

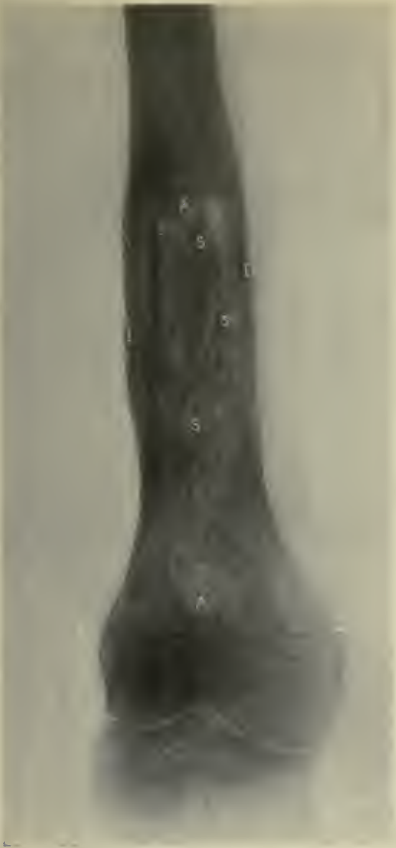


Fig. 46.—Multiple osteomyelitis the result of failure to recognize promptly the primary focus of bone infection. Anteroposterior roentgenogram of left femur before operation. Note that the medullary involvement here has been confined chiefly to the lower half of the shaft. Inside the central abscess cavity (*A A*) lie several sequestra (*S*), the largest being located at the upper end of the cavity. Note the considerable size of the involucrum (*I*) formed and its evident origin from the periosteum by bony apposition between the periosteum and the cortex of the shaft. In spite of the considerable size of the central abscess cavity, the involucrum has strengthened the bone sufficiently to carry safely the weight of the body. Note how abruptly the abscess cavity stops at the lower epiphyseal line (*E–E*) of the femur. Bone phlegmons do not often pass the epiphyseal line, and when they do, are usually caused by the streptococcus, not the staphylococcus.

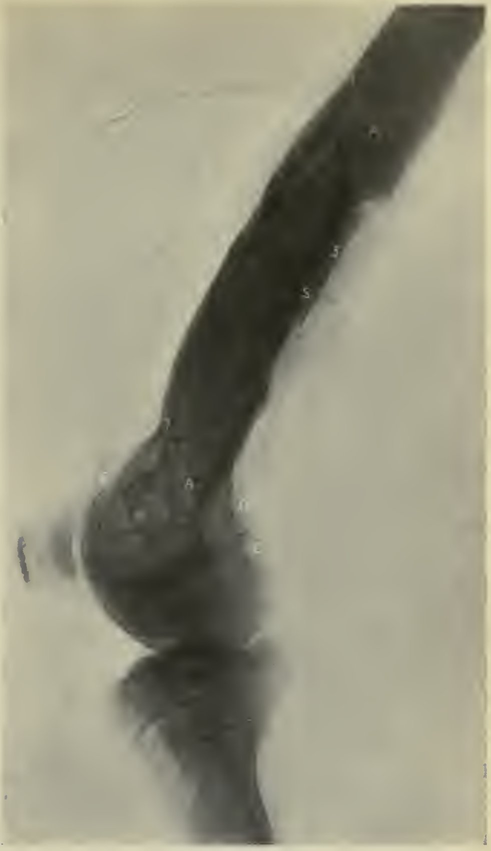


Fig. 47.—Multiple osteomyelitis the result of failure to recognize promptly the primary focus of bone infection. Lateral roentgenogram of left femur before operation. Note the confinement of the medullary involvement to the lower half of the femoral shaft. Note the posterior cortical defect (*D–D*) in which the largest sequestrum lies (*S–S*). Note that the involucrum (*I*) is practically coextensive with the abscess cavity, (*A*), and that both are limited below by the epiphyseal line (*E*). The shadow (*B*) in the soft parts directly behind the posterior femoral defect indicates the extent of abscess formation in the soft parts. This shadow is thrown by particles of bone floating in the pus or lodged on the abscess walls.

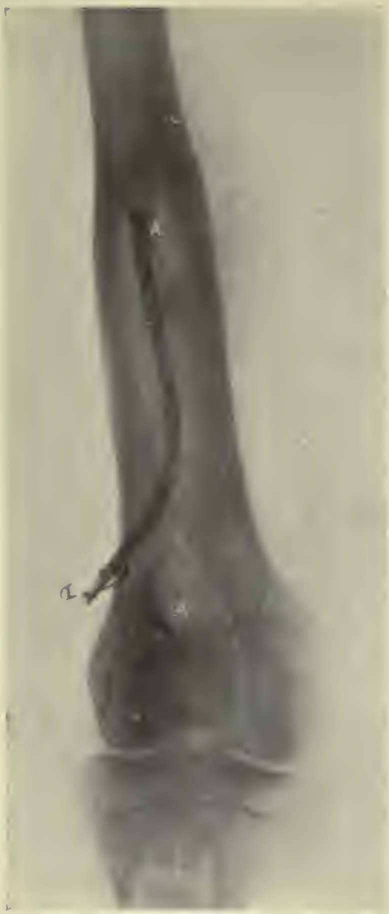


Fig. 48.—Multiple osteomyelitis, the result of failure to recognize promptly the primary focus of bone infection. Antero-posterior roentgenogram of left femur soon after operation. Note how thoroughly the abscess cavity has been opened in order to make certain the removal of all sequestra. Note that the fenestrated drain is placed along the floor of practically the entire cavity, which has been opened from the posterior aspect of the thigh, thus allowing perfect down-hill drainage as the patient lies in bed and also after he gets up and about.

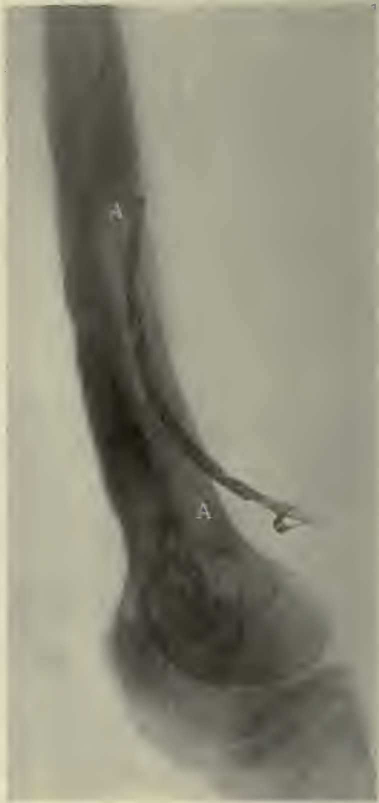


Fig. 49.—Multiple osteomyelitis the result of failure to recognize promptly the primary focus of bone infection. Lateral roentgenogram of left femur soon after operation. Note the thorough opening of the medullary abscess cavity (*A*) from the posterior aspect of the thigh, and easy removal of sequestra and perfect drainage thus secured.

will be left which will require another operation at a subsequent date. Get out *all* the bone at the first operation and cure the patient for once and all.

If eight or ten inches of the shaft is involved in the sequestrum, chisel out a groove in the surrounding periosteal bone of the involucrum eight or ten inches long. Do not try to take out an eight- or ten-inch sequestrum through a seven or seven and a half inch hole. The latter is a very common error, but a costly one—to the patient.

Remember that you can chisel away three-quarters of the thickness of that entire length of bone and not do your patient any material injury. New bone will regenerate to fill the gap.

Fill the cavity which the removal of the sequestrum leaves. Fill it either with skin or a blood-clot or a Moorhof plug; but *do not try to fill it until it is empty.* That statement may sound like a Celticism, but it is meant to convey an important truth. It is no use to fill the cavity unless *all* the dead bone has been removed. All the dead bone *must* come out. If you do not remove it, it will come out finally of its own accord; but in so doing it will certainly bring out your "bone plug" with it.

Like most of the acute infections, osteomyelitis is primarily a problem of the general practitioner, and on the shoulders of the general practitioner rests the chief responsibility as to whether these patients live or perish, or, if they do live, whether they have the usefulness of their limbs preserved for them or not.

The second patient teaches us a lesson equally as instructive as the first, although somewhat different. She is a dispensary patient who has come to us this morning for the first time. We have not had the opportunity yet to study her case as carefully as we have studied this boy whom you have just seen, but the lessons which her case teaches are fairly obvious without having recourse to any laboratory aids except the *x*-ray, and we have already had the necessary roentgenograms made and placed in the shadow-box, where you now see them. Tell us about this patient.

Intern: She cannot speak English. She is Italian—so I have been unable to obtain a history.

Dr. Murphy: Yes. So now you must become a veterinarian, as it were. You must make the diagnosis without information from the patient. That is a fine opportunity for you to show your diagnostic ability. If you were acting the part of a psychiatrist, however, instead of a veterinarian, you would be still worse off. A psychiatrist often must make his physical diagnoses in spite of a false history. Although some classes of insane patients, such as practically all paranoiacs, most maniacs, and many melancholiacs, can give a fairly accurate and reliable history of their physical ills, other classes, such as the senile, the paretics, the hysterics, the alcoholics, the cocainists, and the morphinists, fabricate extensively. Demented and confused patients can give practically no history at all, as a rule, and in such cases the psychiatrist has the advantage—dubious though it may be—of working on the veterinary basis, instead of struggling with falsifications.

You must have an interpreter. Dr. Hochrein, what is the matter with this patient?

Dr. Hochrein: She complains of pain in her ankle.

Dr. Murphy: That is almost the sum-total of the information we have. What is the matter with her? Remember that a woman's ankle is located all the way from her internal malleolus to her knee. What is the matter with her?

Intern: Pain in which ankle?

Dr. Murphy: That is a fine question, doctor. Accuracy counts. Which ankle, Dr. Hochrein?

Dr. Hochrein: The left.

Dr. Murphy: She complains of pain in the left ankle. She certainly does not complain much. [The patient removes her stocking and shows the left leg extensively scarred—especially along the tibia.] I think I was correct in my localization of this woman's ankle, judging from the looks of the leg. What is the matter with her?

Intern: She has had an operation performed there.

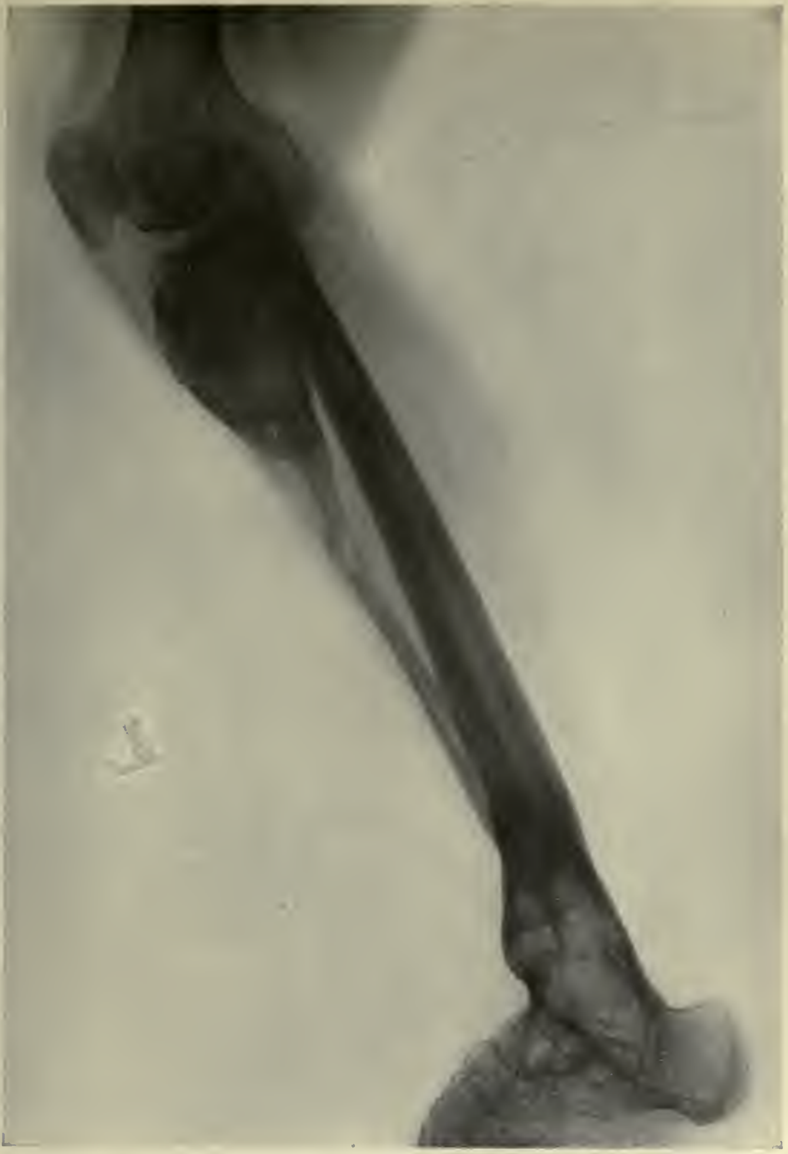


Fig. 50.—The after-result of an improperly managed case of acute osteomyelitis. Lateral roentgenogram of left leg, left knee, and left ankle-joint. Note the tenuous and fractured remnant of the shortened and now useless tibia. Note the upward dislocation of the upper end of the fibula. The extent of the dislocation corresponds with the degree of shortening of the tibia and of the entire leg, viz., about two inches. Note the solid bony ankylosis of the ankle-joint. There is firm bony union between the astragalus and all the bones surrounding it except the scaphoid. Note the marked increase in width and density of the fibula which now transmits all the weight borne by the limb. It is chiefly the downward thrust of the weight of the body in walking which has produced the upward dislocation of the head of the fibula by stretching the ligaments binding the fibula to the tibia (interosseous ligament, anterosuperior and posterosuperior tibiofibular ligaments). Note the atrophy of disuse in the femur, the patella, and the tarsal bones, and how strikingly this atrophy contrasts with the hypertrophy of the overloaded fibula.

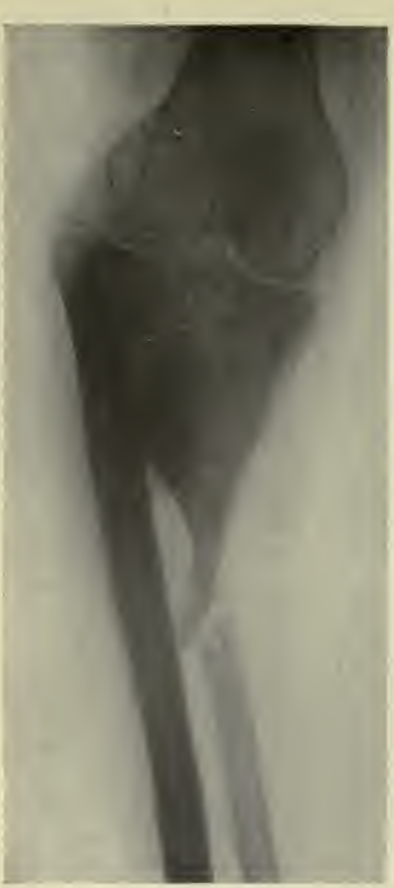


Fig. 51.—The after-result of an improperly managed case of acute osteomyelitis of the left tibia. Anteroposterior roentgenogram of the upper half of the leg and the knee. Note the slender broken and useless remnant of the tibia and the upward dislocation of the fibula. Note the genu varum which has resulted from the change in the direction of the line of thrust of the femur. This thrust is now transmitted outward as well as downward on the fibula. Had the latter bone been transplanted early in the course of the disease under the center of the head of the tibia, as suggested by Dr. Murphy, neither the present shortening of the limb nor the genu varum would have resulted.

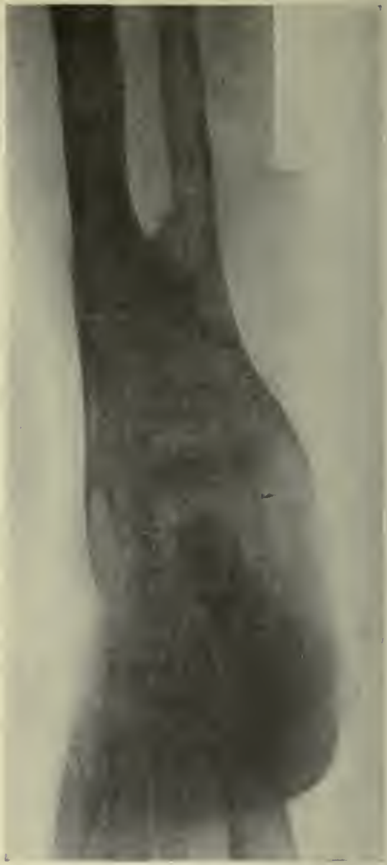


Fig. 52.—The after-result of an improperly managed case of acute osteomyelitis of the left tibia. Anteroposterior roentgenogram of the lower half of the leg and ankle. Note the slenderness of the tibia and the hypertrophy of the fibula which now transmits the body weight. Note the bony ankylosis of the ankle-joint and of the adjacent bones to one another—tibia, fibula, astragalus, and calcaneus. Note also the atrophy of disuse of all these bones except the fibula.

Dr. Murphy: I think so. It certainly looks like it. What was the matter with her?

Intern: Probably there was an osteomyelitis.

Dr. Murphy: Suppose I think it was a tuberculosis.

Intern: We have no history to guide us.

Dr. Murphy: Very well. You see that one is indeed in difficult circumstances when trying to make accurate diagnoses without a history. When I was a student in Vienna my American colleagues and I who congregated mornings in the dead-house had no great regard for the somatic diagnoses submitted with the cadavers from the old *Irrenhaus* in the Lazarethgasse, the city insane asylum, which has rather recently been removed to a magnificent new location at Steinhof, overlooking the Wiener Wald, in order to make the old grounds available for the great modern clinics of the new *Allgemeines Krankenhaus*. We used to see a good many diagnoses, such as: *paralysis progressiva*, *marasmus et myodegeneratio cordis*, or *senile Demenz*, *Arteriosclerose und Cachexie*, or *akute Verwirrung*, *Exhaustio*, *Albuminurie*. At these autopsies pathologic lesions of importance not rarely came to light without their presence being denoted on the diagnosis card accompanying the body; and diagnoses which were seldom overlooked in the other clinics were rather frequently—so we thought—missed at the Irrenhaus. We were ignorant of the conditions under which many of these diagnoses had to be made, namely, that the insane patients most liable to die are the very excited—whom it is difficult or impossible to examine properly—and the greatly demented, from whom it is impossible to elicit a clinical history, and who are often negativistic and resist examination with such strength as they still possess.

In our ignorance of these conditions we often spoke of the "psychiatrist's diagnosis" in a deprecatory manner, which the circumstances in no way justified. It was only when subsequently I was called on to make some of these diagnoses myself under like conditions that my attitude toward the Vienna *Klinik für Psychiatrie* and the *Irrenhaus* changed from one of amused or critical toleration to one of warm admiration. I had no better and, probably, worse luck at making the somatic diagnoses, because in mental diagnosis and prognosis I found myself in a virgin field, where I was a complete stranger, and quite unable to value properly the patient's statements. That is an about-face attitude which we are often forced to assume, when, after criticizing another man's way of handling his problems, we are called on to take up his burden ourselves.

This patient has practically no motion at the ankle between the astragalus and the tibia. The motion which she still possesses here is between the scaphoid and the astragalus. The astragalus seems to be fixed firmly to all the adjacent bones except the scaphoid. What is the matter with her? *Now* it is important to get an interpreter. Dr. Hochrein, tell us the story.

DR. HOCHREIN: On July 23, 1912, she got her feet wet. Two days later she was taken with a chill, pain in her ankle, and a high temperature. On the second day of her illness her doctor made an incision in the soft parts over the ankle, looking for pus, and found nothing but blood. Eight days later he made this extensive longitudinal incision over the tibia and took out a long piece of "dead bone," as he called it. She lay in bed for six months with the wound still discharging. She now has had this shortened leg and has had trouble in walking on it ever since. She comes here to see what can be done.

DR. MURPHY: What was the matter with her? What are you going to do for her?

INTERN: According to the history, it was a case of osteomyelitis.

DR. MURPHY: What makes you think it was an osteomyelitis?

INTERN: The chill and the fever.

DR. MURPHY: No. *First*, the exposure; second, the chill; and third, the pain, where?

INTERN: In her ankle.

DR. MURPHY: No. *Near* the ankle. Then the making of the first incision on what day of the illness?

INTERN: The second day.

DR. MURPHY: And the surgeon found no pus—only some hemorrhage. Finally what?

INTERN: Eight days later he exposed the bone, found pus, and removed a large piece of dead bone.

DR. MURPHY: All right; what is the matter with her? She says the doctor took out what?

INTERN: Dead bone.

DR. MURPHY: What do you think about it?

INTERN: I think that probably the ankle was opened and infected by the first incision and that subsequently it ankylosed.

DR. MURPHY: Only an ankylosis? No, worse than that.

INTERN: The wound discharged pus for a long time.

DR. MURPHY: All right. Tell us about it. What was the matter with her?

INTERN: There was a suppuration of the ankle and a destruction.

DR. MURPHY: We do not know that, doctor. Tell us the things we do know.

INTERN: First an ankylosis of the ankle.

DR. MURPHY: But the leg measures nearly two inches short. Part of the tibia is gone. What has become of the fibula all this time? If only the tibia is two inches short, she had an osteomyelitis of the tibia alone. But since the leg itself is two inches short, something must have happened to both bones. What has happened to the fibula?

INTERN: It was not infected.

DR. MURPHY: What has happened to it if it has allowed the leg to become two inches short? There is certainly not two inches lost in the slight compensatory curve which one sees in the fibula. There is the upper end of the fibula [pointing]. The articular surface of the tibia lies about here, five-eighths of an inch at least below the upper end of the fibula. Let us look at the *x*-ray plate of the knee. It will save time. (See the accompanying anteroposterior roentgenogram of the left knee.) The fibula, instead of lying half an inch *below*, has passed above the upper articular surface of the tibia. The shaft of the tibia is thinner than the shaft of the fibula, and, a few inches below the tuberosity, its continuity is interrupted, which does not mean necessarily that it has been fractured. This interruption may be the result of necrosis following the original inflammation. Note the hypertrophy that has taken place in the fibula to help to carry the body burden. Note in the x-ray plate the thickness of the fibular cortex and its density.

Let us see how this evil result could have been avoided. Let us see what contributed to this condition. Let us see the rôle that should have been played by the doctor. The case is a classic one of osteomyelitis with the typical history of exposure and the chill. The precipitation of the infectious organisms in the lower end of the tibia produced an acute osteomyelitis, which the doctor probably diagnosed. He had just nerve enough to open the skin and open it early; but then he became frightened. He did not cut down to the bone and then drill open the marrow, as he should have done. We know that in acute suppurative osteomyelitis there is no pus outside the bone, as a rule, in the twenty-four or forty-eight hours of the disease. During that period the pus remains within the bone. If the doctor had just taken a gimlet and drilled a hole in the bone, he would have let the pus out of the medulla and would have saved the shaft from destruction. If he had cut down far enough, that is, down to the bone, he would have seen the edematous periosteum, and would have known that the pus lay in the marrow beneath, because the periosteum is always edematous, even very early, in acute suppurative osteomyelitis, and this edema is the surgical guide to the pus beneath.

That neglect to cut deep enough was error number one in the doctor's management. Then came error number two. You have heard me speak often of the failure of the average surgeon to make a sufficiently large incision to get out all the dead bone in an acute osteomyelitis. But by that statement I mean *when the bone is dead and separated as a sequestrum.* Dead bone never separates as a sequestrum in eight days. Living bone and dead bone do not pull apart one from the other and separate like living and dead soft tissues. The separation is due to the action of the living bone in producing an absorption of bone and development of granulations at the line of demarcation; and that process takes from four weeks to six or eight or, more likely, even ten weeks to complete itself, the time required depending largely on whether or not the line of demarcation remains stationary. If the line of demarcation advances, the time required for separation is just so much longer. When this patient's doctor operated at the end of eight days, what did he do? In endeavoring to perform a complete operation he cut away most of the shaft before the sequestrum had separated and before he knew how much of it was going to die and how much of it live.

The indication in the management of this case in the beginning was what? To make a skin incision the full length of the tibia, if necessary, for exploration of the surface of the bone; then to trephine the bone where the suppurating focus was located and let the pus out. Usually a single small puncture through the cortex is all that is necessary to let the pus escape. It does no particular harm to make more openings if one wishes. But the operation, whatever it be, must be performed in twenty-four or thirty-six hours from the onset of the pain if necrosis of the shaft is to be avoided. It is the danger of extensive necrosis, as well as of pyemia, which compels us to do the early operation. This patient's doctor had one correct idea—the necessity of early action; but he did not have positive convictions in his cortical cells that pus lay within the bone, and not on its surface. He rather suspected a phlegmon or a periosteal suppuration when he should have known that an acute primary suppurative periostitis is a rare affection. I doubt if I have seen five in all my experience. Acute suppurative periostitis occurs occasionally with gonorrhea, and is occasionally metastatic in typhoid or scarlet fever; but it is an extremely rare lesion, while an acute metastatic central osteomyelitis is one of the common lesions of bone, and by direct spread of the suppuration along the bone canals to the surface is by far the most frequent cause of suppurative periostitis.

You may have noted that in both these cases which I am showing you to-day it was the tibia which was primarily involved. The tibia is, in my experience at least, the bone most frequently affected by osteomyelitis. The femur comes next in order. Perhaps these two long bones are the most frequently infected, because of the large area which they offer for infection, that is, on account of their size, and because growth in their epiphyses is more active than in the epiphyses of any of the other bones; for it is a general rule that osteomyelitis tends to localize near those epiphyses which grow most actively.

Next in order of frequency come the other long bones. Infection of the short and flat bones is much less frequent; but the clavicle, scapula, ilium, os calcis, ribs, and skull are occasionally the sites of metastatic infection. The vertebræ are still more rarely the seat of an acute or subacute suppurative osteomyelitis, although we have had a case of it within the last year. Donath has made a thorough study of this interesting variety of osteomyelitis, which you will find in von Langenbeck's Archiv for 1906 (*Archiv für klinische Chirurgie*, vol. lxxix).

Because osteomyelitis of the flat bones is a rather rare affection is no excuse, however, for overlooking the condition when it presents itself. Such an oversight is just as fatal an error as in the case of osteomyelitis of the long bones. An instance occurred recently in the practice of a young surgical friend of mine. The case was such an instructive comedy of errors, or, rather, tragedy of errors, that it well deserves to be related in this connection.

The patient, a youngster of eight or nine years, fell through the ice into the water a week or ten days after he had had an acute coryza and pharyngitis, accompanied by an abundant herpes. His clothes were soaked through by the icy water. He was thoroughly chilled, and his teeth were chattering when he reached home. He was put to bed—without a hot bath—and went to school the next morning. He came home at noon complaining of pain in his right side; and so his aunt—his mother is dead—kept him home from school in the afternoon.

In the course of the afternoon he had a chill, and by night the pain was so much worse that a doctor was sent for. He promptly diagnosed appendicitis—the patient had some fever and slight tenderness and slight rigidity low down on the right side—and *put the boy on "expectant treatment"*! Whatever chance the boy had for his life was probably lost right there by the doctor's failure to make the correct diagnosis, and his equally serious failure to institute the proper treatment for the condition he diagnosed—*immediate operation.* Had he operated immediately he would have found nothing the matter with the appendix, and, recognizing his diagnostic error, would have searched elsewhere for the cause of the trouble, and would, very possibly, have found it.

There was, however, very little justification for the diagnosis of appendicitis. Appendicitis does not begin with a chill. It begins with diffuse abdominal pain, followed immediately by nausea, vomiting, and fever, and, subsequently, by gradual localization of the pain, tenderness, and rigidity to the right lower quadrant of the abdomen.

Following the patient's chill there was no nausea or vomiting; but the initial pain in the right side became much worse and fever appeared. Moreover, the history of a nasal infection, accompanied by a well-marked herpes and followed by a fall through the ice and a thorough chilling one week later, on the day before the development of his acute symptoms, is typical for an osteomyelitis and unusual for appendicitis. A metastatic diffuse peritonitis may follow an acute rhinitis or pharyngitis, but an appendicitis rarely does. A history of constipation or digestive disorders is much more frequent with the latter affection.

The patient was treated for two days on the "expectant plan." I call it the "*expectans mortem* treatment," because death is about all the patient has to expect when this line of treatment is carried to its logical conclusion and is not interrupted by an operation.

The patient had a chill or two each day, and grew rapidly and progressively worse. The family insisted then that something be done, and so the patient was sent to the hospital. Three other doctors then saw him, all of whom concurred in the diagnosis of acute appendicitis. A white count was made, which showed 30,000 leukocytes! The diagnosis, accordingly, being verified (!), the surgeon was called in to remove the appendix.

The surgeon recognized the case at once as one of acute osteomyelitis, and not of appendicitis; but he made the error of considering it an osteomyelitis of the neck or upper shaft of the femur. There was slight swelling of the upper right thigh, which, just below the trochanter, measured one inch more in circumference than the left. The right trochanter could not be quite so clearly outlined on palpation as the left, because of the slight edema around it. The patient could not lift the right leg off the table on account of the pain in the region of the hip, whereas the left leg was moved with normal freedom.

The possibility of osteomyelitis of the ilium was not seriously considered because of its rarity. If the surgeon had carefully palpated the ilium, using deep pressure, he would have located the focus. The surgeon did note that there was hyperesthesia over the ilium, but he thought it was the same superficial hypersensitiveness which the frightened youngster showed in the upper thigh and the lower abdomen. He also noted that there was no tenderness on deep pressure over the great trochanter and the femur below the trochanter. Therefore he erroneously concluded that the pus must be in the neck of the femur.

Accordingly, the boy was anesthetized and one hole was drilled in the neck of the femur and then a second slightly larger one. Neither "tap" drew pus, even after some waiting. Was it a small pus focus which had not been reached by either drill opening, but which would open into one or the other of them as it spread—perhaps in a few hours? The surgeon thought so. The other consultants thought not. They still believed it an appendicitis. Therefore the abdomen was opened, and a long appendix with a concretion at the very tip was removed. The appendix showed no signs of inflammation outside or inside. The exploration of the abdominal cavity on the right side revealed nothing abnormal.

Consequently, before proceeding further, it was decided to wait until more symptoms developed, and the region between the great trochanter and McBurney's point was examined twice daily *but not by deep pressure*—chiefly by the eye and by superficial palpation, because of the patient's growing hypersensitiveness. Another surgeon was called to see the case, and he too failed to use deep pressure palpation and so failed to make the diagnosis.

In forty-eight hours noticeable swelling appeared a little below the crest of the right ilium, followed by redness. The diagnosis then was certain, but it was too late. The abscess was opened and evacuated, and found to originate from an osteomyelitis focus in the right ilium somewhat above the acetabular cavity. The cortical bone was chipped away to permit free drainage, and a tube drain inserted.

The drainage of the abscess produced a temporary improvement in the patient's condition, but was performed too late to prevent pyemia.

The first pyemic focus developed in the left elbow-joint, and others appeared rapidly over the body. The little patient hovered between life and death for two weeks; then came a suppurative pericarditis and exitus.

It was an instructive case, but a very sad one, because a useful young life was cut off so unnecessarily. Perhaps it is the rule that our most instructive cases are our saddest. It seems to be human nature to learn more by failure than by success. The case emphasizes all too well the overwhelming importance of accurate diagnosis in acute surgical conditions. Diagnosis is far more important here than is surgical technic, and it seems to be the last thing the surgeon learns, whereas it should be the first.

It is not always easy in an individual case to determine whether the primary seat of a bone suppuration is the cortex, the marrow, or the periosteum. A medullary infection spreads rapidly to the outside of the bone along the cortical canals. A cortical infection, an osteitis, may spread along the same channels in both directions; and the pus from a suppurative periostitis may follow the same path to the marrow that medullary pus does to the outside of the bone. By far the most frequent route, however, is from within outward.

The therapeutic rule which I have laid down holds good, however, for all three varieties of infection. When you find pus beneath the periosteum, look for it also in the medullary cavity. You will practically always find it there. But even if you should not find it there in an individual case you will have done the patient no harm by the mere trephining of the bone, and will have eliminated a source of worry for yourself. It is better to do such a procedure a dozen times unnecessarily than to neglect it once when it is needed.

When the microörganisms present are very virulent, the periosteum may become separated from the bone over very extensive areas. Although such a periostitis dissecans may give rise to severe and extensive phlegmons of the soft parts, it does not produce a total necrosis of the shaft of the bone unless there is an accompanying osteomyelitis—which there is, of course, as a rule. Instead of a total destruction, a periostitis, at the most, produces only a very superficial necrosis of the bone, a mere exfoliation.

When necrosis of the shaft occurs, the dead bone at first presents the pink whiteness and smoothness of normal bone. It is only when suppuration and granulations in the cortical canals have produced defects in the dead bone that the latter comes to appear gnawed out and discolored.

I hope I have impressed upon you the intimate relation between the pathologic changes present in acute osteomyelitis and the symptoms and signs produced thereby. I have not gone into the subject of the pathology and diagnosis of the rarer forms of acute periostitis, osteitis, and osteomyelitis, which is a fascinating but extensive chapter in surgery, because I want to drive home the practical clinical lessons which these two clinical cases teach.

The second error in the doctor's treatment of this woman was that he endeavored to remove the dead bone eight days after the beginning of the infection. He should merely have given vent to the pus present and then should have waited—several weeks, if necessary—until the sequestrum separated from the living bone. Not until then should he have attempted a sequestrectomy. By that time a solid involucrum would have formed, and no shortening of the tibia would have resulted.

So long as he actually did endeavor to remove the bone at that time, however, what else could he have done to avoid the subsequent shortening? He could have cut off the upper articular end of the fibula and have transplanted the fibula squarely under the middle of the upper end of the tibia; that is, in the midline. In place of the deviation and shortening of the limb which the patient has now, she would have developed a straight leg of normal length. The pressure on the fibula from the weight of the body and the pull of the muscles would have developed the bone to a size sufficient to support the body.

The patient also has a bony ankylosis of her ankle. Why? Because in taking out the bone the doctor stumbled into the ankle-joint and allowed pus to enter and infect it. Remember that osteomyelitis practically never destroys the bone beyond the epiphyseal line, except occasionally in virulent streptococcus infections. Remember that the epiphysis is the *trocha* at which the osteal phlegmon usually stops. If the infection is virulent enough to attack the epiphyseal cartilage extensively, epiphyseal separation will usually occur before epiphysitis develops.

This second operation was meddlesome surgery, and has given this patient a stiff ankle and a shortened, useless leg. With proper surgery she should have had a perfect anatomic and a perfect functional result. Of course, even with this crude surgery it is quite possible that the doctor saved the patient's life. Although he let out the pus late, that is, on the eighth day, *he let it out*, and this drainage may have saved the patient from developing a pyemia from this severe infection. Of course, she does not realize the perils which she missed. She realizes only the fact that she must go through life with a useless leg. We know that the surgeon at first was too conservative to be effective, and later too radical to be safe; but we also know that his poor surgery was better than none at all, in that it was life-saving. To this last fact the average patient pays no attention, and this woman is not at all above the average. Her mind is entirely centered on the present condition of her leg. Imagine what harm such a patient could do a doctor's practice in a small town, especially if there were other osteomyelitis cases in town who had received *proper* treatment! Imagine the comments which the difference in the results would occasion!

This case could scarcely be better designed for clinical teaching if one tried to produce it artificially. It shows so strikingly the results of some of the common errors in the management of acute suppurative osteomyelitis. It illustrates, first, the proper time for opening an osteomyelitis focus and the result of not opening it at the proper time; second, that, when opening the focus, one should do nothing more than simply give vent to the pus; third, that the time for the removal of dead bone is not immediately after the necrosis has occurred, but some weeks later, when the resulting sequestrum has fully separated. This case demonstrates forcibly the evil result of not following this well-established surgical principle. Fourth, this patient illustrates the fact that, when total or nearly total necrosis of the tibia occurs from an acute osteomyelitis, the loss of use of the leg through shortening and lack of bone support may be prevented by using the fibula to replace the tibia.

Let me show you the photograph and *x*-ray prints of a similar case in a growing child who suffered a nearly total destruction of the tibia very much like the destruction in this case, and also accompanied by a sliding of the upper end of the fibula past the head of the tibia and above the articular line of the knee-joint. We first saw the case when it was still actively suppurating and discharging pus through several sinuses. After the major portion of the sequestrum had been removed and suppuration was still enveloping the entire length of the bone, we transplanted the fibula, in the manner which we have just described. We planted the cut-off upper end of the fibula against the lower portion of the upper tibial epiphysis. Then we kept the patient at rest in bed while the bone was healing in place and while the suppuration was being combatted. The contracture of the leg muscles which always takes place when this operation is not performed was thereby prevented. When the discharge had ceased and the wound had fully healed, we inserted a long transplant of bone from the other tibia to assist the fibula in supporting the limb. The result is that the little patient now has a leg of normal length, in which the fibula and the tibial transplant have developed to additional size, so that the patient walks without the slightest limp. If you wish to realize fully how important such a result is to the patient, just imagine that she were your only child, a daughter. Think what a useful limb means to her future happiness and welfare! You should think of these patients not as cases number one, two, or twenty-two, but as other human beings, the treasures of devoted parents. Then you can feel properly the obligation that goes with the care of such a case.

[NOTE.—The above-mentioned operation, consisting of transplantation of the fibula with subsequent transplantation of bone from the other tibia, was recommended to the patient. She did not wait to hear all the details of the measures proposed. At the word "operation" she picked up her things and made a rapid exit. Her experience, apparently, has led her to fear operation more than she fears disease; and perhaps this attitude has some justification in her case. Up to the present time, March 1, 1915, she has not returned for further observation.—ED.]

RECURRING MULTIPLE OSTEOMYELITIS AND PERIOSTITIS. CURETMENT OF SINUSES AND GRANULATION MASSES

HISTORY

THE patient, a female aged twenty-nine years, entered the hospital November 27, 1911, because of pains in and enlargements of the bones of her arms and legs. Her father died at the age of eighty-five years of acute pneumonia. There is no tuberculosis or carcinoma in her family. Her past personal history is negative. Her present trouble started as follows:

In October, 1894, three weeks after an attack of influenza, the patient experienced a pain in the left ankle, so severe that she had to go to bed. The following day she felt very ill, was nauseated, and had a chill, fever, and sweated profusely. The ankle commenced to swell immediately and the skin over it became red. The family physician poulticed the ankle and administered medicine for "rheumatism." Three weeks later he "lanced the ankle" and a discharge of several ounces of pus followed immediately on lancing. The pus daily continued to drain from the ankle. Seven weeks after the onset of the affection the patient was taken to a hospital, where the ankle-joint was cureted and several pieces of dead bone and a great deal of pus removed.

During the next five months the ankle was operated four times, loose pieces of bone being removed at each operation. The ankle continued to discharge for a year and a half after the onset of the trouble. During this time the sinus leading into the ankle healed up and broke open again five times. The patient was not recovered sufficiently to use crutches until one year after the beginning of her illness.

Four months following the onset of the lesion in the left ankle the right ankle commenced to hurt when she put her weight on it. This pain was not severe at first, but the ankle soon began to swell and the skin over it became red. At this time her temperature, which was already above normal because of the infection in her left ankle, went up still further. The right ankle was aspirated and fluid removed from it one month after it first began to cause pain. It was again completely free from pain in two months after the aspiration, but did not remain well, and had to be operated twice in the next two years. Then it healed and gave no further trouble until 1908, when it again became tender and swollen, and the skin over it red; but this mild attack cleared up spontaneously in two weeks' time. Six times during the last three years the right ankle has become swollen and red. Each time the swelling and redness have cleared up in from one to two weeks under local palliative treatment. The ankle has not been operated since 1896.

Twelve years ago (1899), five years after the original osteal infection, the right and left shoulders began to swell and cause the patient pain. One year and a half later they "broke down," discharging small particles of bone and pus. The sinuses healed up entirely only at the end of three years. During that period they had repeatedly broken open, discharged pus, and then healed up, only to break down again.

Four years ago (1907) she had several glands just inferior to the right clavicle removed. The physician who removed these glands said they were tuberculous. In 1902 she had the lower portion of her right forearm operated on for what at that time was diagnosed as "tuberculous abscesses." In 1905 and 1908 similar operations were again performed at the site of the incision made in 1902. These operations were for a recurrence of the "tuberculous abscesses."

The patient was operated at Mercy Hospital, December 2, 1911. A thickened periosteum over two granulating foci in the right tibia was found. These two foci were opened, cureted, and closed without drainage. Primary union resulted. The periosteum of the lower portion of the right tibia was found similarly involved and was similarly treated. The wound then healed uneventfully.

COMMENTS AND OPERATION

Dr. Murphy (June 18, 1914): Did the patient injure the left ankle in any way?

Intern: The history does not state.

Dr. Murphy: What is the matter with her?

Intern: She had a chronic osteomyelitis.

Dr. Murphy: Read the last sentence in that history.

Intern: "These operations were for a recurrence of the tuberculous abscesses." She comes here now because she is still having trouble with these old suppurating foci. She has suffered no recent injury.

Dr. Murphy: Go back to the beginning of the patient's history. What was the matter with her in the beginning? When we decide that point, we can very likely tell what is the matter with her now.

Intern: She had a lesion which began as a swelling of the left ankle. An abscess then formed which opened spontaneously and discharged pus.

Dr. Murphy: That sequence is common with tuberculosis, is it not? Therefore, what is the matter with the patient?

Intern: A tuberculous osteomyelitis.

Dr. Murphy: Why?

Intern: I do not know.

Dr. Murphy: If you do not know, why do you state so positively what is the matter with her? What do you *think* is the matter with her?

Intern: She has an osteomyelitis of some kind.

Dr. Murphy: Why not a tuberculous osteomyelitis? All the diagnostic points you have mentioned are quite usual in tuberculosis.

Intern: The discharge of bone and pus from a focus in the bone showed that this must have been an osteomyelitis.

Dr. Murphy: I have a patient with osteal tuberculosis living in Los Angeles, California, from whose leg I took out 20 pieces of bone, as well as a large amount of pus. What have you to say to that fact? There was no doubt about the diagnosis, because it was verified by bacteriologic as well as histologic examination.

Intern: This patient has no history of an injury.

Dr. Murphy: It is not necessary to receive an injury in order to develop bone tuberculosis; in fact, tuberculosis practically never develops at a fracture site, nor does a sarcoma. Your answers are less apropos every minute. Why? Because you have no clean-cut idea of the clinical course of a bone tuberculosis or of a septic osteomyelitis. From this patient's history, as you have read it, one could make a diagnosis one hundred years from now, if necessary. It is characteristic and typical of the condition. Many of the finer details of the patient's story are missing, but the salient points are there.

In the first place, the onset of this trouble was sudden. Bone tuberculosis never incapacitates a patient in a day, or two days, or three days, or five days, or ten days—*absolutely never*, I believe I am perfectly safe in saying. The patients never develop a chill or a high temperature in the initial stage of an osteal tuberculosis. Judging from the mode of onset of this affection, with severe local pain, a chill, and a high temperature, it could have been only one of two diseases. Either she had a metastatic arthritis of the ankle or a metastatic osteomyelitis of the lower tibial metaphysis. *It could not have been a tuberculosis.* Never mind how many sinuses she has or has had—and she has them now in many bones all over the body. They are simply the results of subsequent similar metastases in the bones, that is all.

She was in bed and completely incapacitated within twenty-four hours after the onset of her original trouble. True, she has no injury that she can now recall; but the probability is that she had some slight trauma, strained or twisted or struck her ankle, but did not pay any attention to the slight mishap, since it caused her no immediate inconvenience. But the microörganisms circulating in her blood probably from her grip, which was in its acute stage three weeks earlier, thus found a *locus minoris resistentiæ* as a nidus for their growth, and thus started the trouble from which the patient is still suffering.

Many of these cases of acute osteomyelitis, in fact, most of them, are diagnosed "rheumatism" by the family physician when first seen, and are so treated. This is a disastrous mistake, and yet I presume that it is made in general practice in close to 19 out of every 20 cases. It is almost as easy to make a careful examination and establish an accurate diagnosis in every case of articular and periarticular lesions as it is to pat the patient on the head and tell her that her pain is "rheumatic," without troubling her to remove her stocking. It is a very strange and very lamentable fact that so few family physicians recognize the urgent necessity of careful physical diagnosis of every case that they see in their practice.

"Rheumatism" serves as a blanket for many sins in diagnosis. Here it covered a capital crime. The correct diagnosis could have been made in this case on the very day of onset. How? By noting that the pain was periarticular, not intra-articular. The local sensitiveness was just proximal to the joint—not in it. *And the joint was free from effusion.* In a metastatic arthritis of the ankle, which in its onset may somewhat resemble an acute osteomyelitis at the lower end of the tibia, there is a fullness in front of the ankle, just anterior to the malleoli, due to the effusion into the ankle-joint. This fullness is missing early in the course of an acute osteomyelitis. It does not occur until late in the disease, and then only when the pus burrows its way out of the bone into the joint and a secondary arthritis develops.

Instead of making the diagnosis at once and promptly opening the focus of pus, the doctor poulticed the ankle. He thus "ripened" the lesion and let it become large and juicy, instead of plucking it when "green" and small. The focus of infection widened and expanded and a big abscess developed.

That doctor's knowledge of the management of osteomyelitis was in about the same stage of development that our knowledge of the management of appendicitis was in the late eighties of the last century. That was the time when a surgeon would go out of the amphitheater after operating for appendicitis and say to the patient's friends, "Why, I just took out a hatful of pus," and he would be proud of the fact. Now he should say, "I got the appendix out just in time. It was nearly ready to burst," for if he finds pus free in the peritoneum to-day he knows that somebody is to blame for it. The presence of free pus at an appendicitis operation now means nothing less than serious mismanagement. It means that either the patient, the family physician, or the surgeon has grossly mishandled the case. Not only is this fact recognized by our profession, but it is a matter of common knowledge, which many intelligent laymen possess. If the surgeon drains a hatful of pus from a case of appendicitis nowadays, that fact signifies to all concerned that the case has been badly conducted. The same statement holds with osteomyelitis. The formation of a big abscess and a large sequestrum can be avoided. If they are not avoided, the fault may be the patient's for refusing early operation and not the doctor's; *but it is somebody's fault!*

When the local pain over the metaphysis of a long bone is ushered in with a chill and an elevation of temperature, and superficial inspection of the parts reveals no apparent change, does it mean that there is not an osteomyelitis present? No, because this particular suppuration lies inside the inelastic wall of the tibia, if the tibia be the bone involved, as in our case of to-day, and it makes its presence evident on the outside of the limb only in the course of time. Before any cutaneous edema or redness appears in such a case, if you will take the patient's ankle in your hands and press your thumb firmly and steadily over the painful area of the tibia for ten, twenty, or thirty seconds, the patient will fairly scream with the pain of this continued pressure if the lesion is an osteomyelitis or a periostitis. That sign makes the diagnosis. An acute arthritis, a thrombophlebitis incipiens, and even a beginning erysipelas, erysipeloid, or a phlegmon, show no such marked increase of local sensitiveness on deep pressure. In acute osteomyelitis the cortical canals rapidly conduct the infection from the medullary cavity to the subperiosteal zone, and this marked tenderness on continued pressure is the indication of a beginning periostitis, whether the periostitis is uncomplicated or accompanies an osteomyelitis or a more superficial osteitis.

Once the diagnosis is made, what is there to wait for? Every hour's delay results in more bone destruction. The physician should handle this condition as the dentist does. Go to the dentist with a pain in the root of a tooth, and what does he do? He taps it to elicit local sensitiveness. He presses deeply against the periosteum covering the alveolar process to see if a local periostitis is developing. Then he tries the effect of hot and cold water on the tooth. The former increases the pain and the latter diminishes it. Why? Because most dental root infections are due to gas-producing anaërobes and hot water puts the gas present under increased tension. This last test is nearly always negative in osteomyelitis of the long bones because the pyogenic aërobes are the causative factors here and they produce no gas.

As soon as the diagnosis of root abscess is made the dentist drills open the tooth and gives vent to the pus. That is all one needs to do with any osteomyelitis to avoid destruction of the bone; but one must do it well inside of the first forty-eight hours after the onset of the trouble to get good results, because the major portion of the bone destruction occurs within the first forty-eight hours of an osteomyelitis. The marrow cavity *should* be opened within twenty-four hours after the onset, and it *can* be opened in twenty-four hours, for the diagnosis can readily be made this early if only the lesion is suspected and the proper examination made.

Once the diagnosis is established, the operation itself is simple. If you have only your pocket-case with you, that is all you need because wherever you may be when you are called on to treat the case a screw-driver or a gimlet can be borrowed and can be boiled. Locate the focus of infection and drill a hole in the bone over it with whatever tool is handy. If you are over the osteomyelitis focus, the deep tissues will be found edematous as you cut down on them. When you have drilled the hole and taken out the gimlet, you will usually have a "dry tap" at first, and you may say to yourself, "I was mistaken." No, you were not mistaken. Wait a minute and slowly the pus will commence to ooze out through the opening. It will not gush out immediately, as it does on opening a subcutaneous abscess, because the wall of the bone abscess is inelastic and does not collapse. You have to wait until the medullary capillaries which have been under pressure refill and thus force out a few drops of pus. Then you can pack the wound loosely, apply a dressing, and go home. The patient is relieved of his pain, and necrosis of a large area of bone will not ensue. The patient may develop other osteomyelitic or metastatic foci if you fail to find and remove the primary focus of his trouble and microörganisms continue circulating in his blood. But the above line of treatment will always relieve the local condition for which you operated and usually will be followed by prompt and complete recovery.

I will relate an incident which illustrates the superlative degree of inefficient treatment of acute osteomyelitis. At one of the meetings of the American Surgical Association—I think it was the second or third that I attended—a prominent surgeon, now deceased, brought to that meeting seven or eight necrotic shafts of bones that he had removed for osteomyelitis. He thought that the specimens from these extensive operations were evidence of surgical skill. They were just the contrary. They were the trophies of the incompetency of the treatment of these cases in their acute stage. Such extensive necroses are always avoidable by doing the proper thing at the right time, venting that little infection early, and stopping further bone destruction. It can be done, without any hazard, in any house where there is a fire and one can get hot water, and it *should* be done. You do not have to wait three weeks and poultice the limb with Denver mud before opening the focus. By so doing you will only get a larger quantity of pus when you finally operate. *Operate it at*

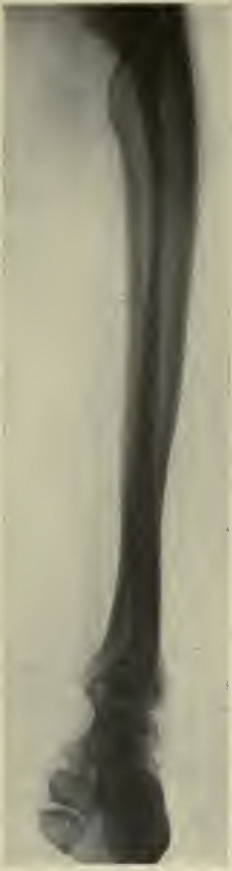


Fig. 53.—Recurring multiple osteomyelitis and periostitis. The lesion here—the cause of the production of the rather extensive subperiosteal granulation masses—was evidently a recurring periostitis and not an osteomyelitis, since the bones themselves appear intact. The slight lipping of the bones making up the right wrist and carpal joints, due to the mild chronic arthritis present, cannot be distinguished in this lateral roentgenogram because of the overlapping of the bone outlines.

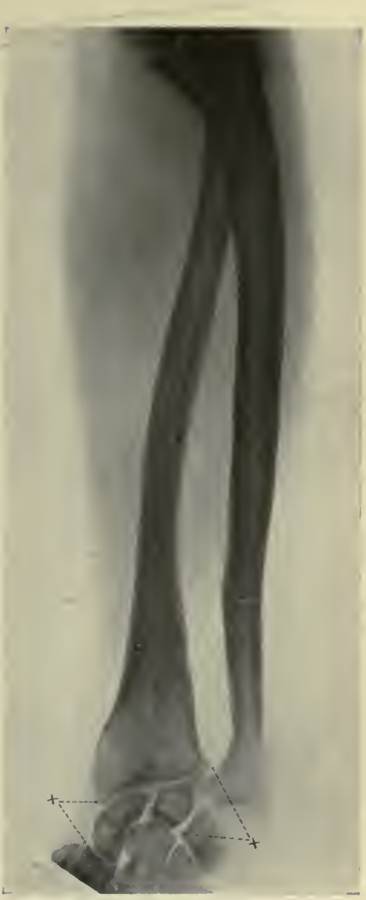


Fig. 54.—Recurring multiple osteomyelitis and periostitis. The subperiosteal granulation masses over the lower metaphysis of the right radius do not show on the *x*-ray plate. The bone itself is evidently not involved. Note the mild chronic arthritic changes at the lower end of the radius and in the carpus, as evidenced by the slight "lipping" (X). Anteroposterior roentgenogram made before operation.

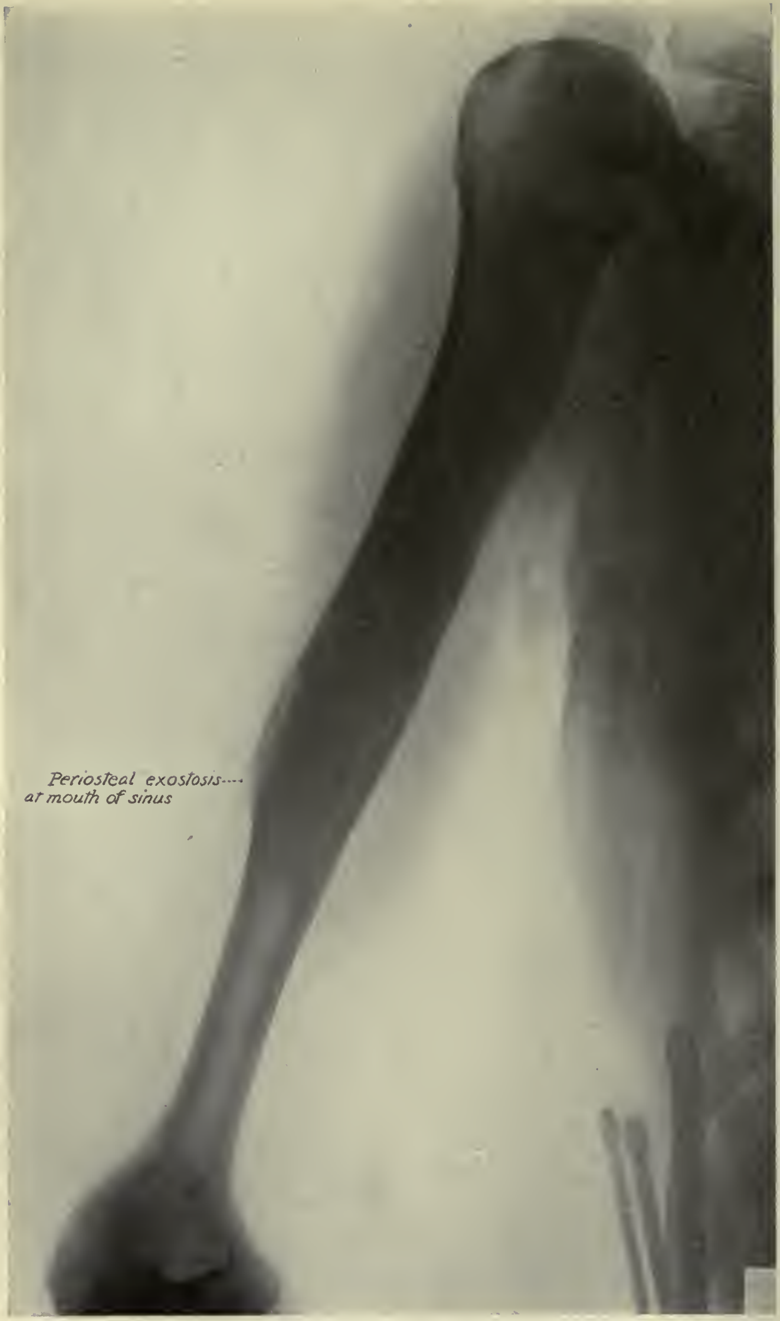


Fig. 55.—Multiple osteomyelitis and periostitis. Old osteomyelitis of the left humerus, with a small fistula running down into the medullary cavity. Subperiosteal granulation mass and periostitic exostosis. Cortical opposition of new bone (ossifying periostitis). Anteroposterior roentgenogram before operation.

once! This imperative statement is not a reflection on Denver mud, but on slipshod diagnosis and tardy surgical treatment.

I am cutting down over the lower metaphysis of the patient's right radius. There appear to be a number of subperiosteal accumulations of granulation tissue here, but none of the foci leads down through the cortex to the medulla. The bone itself is just as intact as the *x*-ray plate indicates. We feel quite certain that there is no tuberculous element to deal with in this girl's case, because we gave her an injection of a milligram of Koch's old tuberculin a few days ago and she showed no local or constitutional reaction.

There appear to be a number of these superficial subperiosteal accumulations on different bones of this girl's skeleton. Her left humerus and right tibia seem to be the only bones which have suffered a medullary involvement. These various low-grade infections keep recurring and recurring. Almost every few months the patient suffers a slight flare-up somewhere.

The tendency for these infections to recur locally is sometimes remarkable. We had one case where the recurrence of the infection did not take place until twenty-seven years after the original osteomyelitis. I operated on the patient myself both times. I call it a recurrence rather than a fresh infection, because a new metastatic suppuration would be much more likely to pick out the exposed metaphysis of one of the numerous other bones in the body than an avascular, eburnated old osteomyelitic relic in which it is difficult for a metastatic infection to penetrate and equally difficult for it to grow after penetration. Many times when operating old osteomyelitis foci one will run across encapsulated sequestra. Where such sequestra are present, recurrences of the inflammation are particularly apt to occur. To the presence of more or less capsulated bacteria remaining from the old suppuration is added the constant irritation of a foreign body—the sequestrum.

We must next open up the old osteomyelitis focus in this patient's left humerus. The palpable nodule near the middle of the bone is an exostosis. This is the sinus which discharged last. It seems to have come from a superficial pocket. The sinuses in such cases, especially when the infection is of a low grade of virulence, may run a considerable distance through the soft tissues before they open to the exterior. The periosteum is not edematous here, and therefore there is no active inflammatory process present. There is a typical granulation mass here beneath the periosteum, and the bone is eburnated; but there is no evidence of any still active infection and no pus. Such a small granulating focus may keep up a prolonged irritation in the tissues and form a nidus for bacterial propagation. Then, when the patient's resistance to infection becomes diminished, for one reason or another, the bacterial virulence may flare up and result in a small infection. There seems to be a small sinus running down through the cortex to the medulla just below this granulation mass. Its walls are solid on all sides. There is no caries. But I am chiseling it clean just the same. Its presence is indicated on the *x*-ray plate just as we found it here. (See the accompanying roentgenogram of the humerus.)

In the days before we had *x*-ray plates we were able at operation to locate these small cavities in about the same way that we do now by the "exostosis of pus exit" which they produce on the surface of the bone, owing to the recurrent attacks of ossifying periostitis to which they are subject.

The patient's right ankle is incapacitated entirely by the old inflammation of the joint. The astragalus moves on the scaphoid, but does not move on the tibia. She has had a number of removals of bone here. Just above the left ankle an incision has been made over the tibia. The infection here did not enter the ankle-joint itself, since motion in the joint is still normal in range.

[NOTE.—The operated foci healed promptly, but the patient will probably develop other foci eventually, as is so characteristic of subacute osteomyelitis of the multiple type.—ED.]

ACUTE OSTEOMYELITIS OF THE RIGHT RADIUS.—INCISION AND DRAINAGE

HISTORY

The patient, male, single, aged seventeen, entered the hospital September 22, 1914. Six weeks ago on a Tuesday night (August 11, 1914) he noticed a scratch on the dorsal portion of the big toe of his left foot. The next day the toe was swollen, but only slightly painful. The patient could not draw on his shoe on account of the swelling. He did not attempt to walk. He had no chill. He does not know whether he had fever. The pain was not acute, unless pressure was applied directly to the dorsum of the great toe.

By the fourth day after the onset the swelling had extended above the left ankle. A doctor who was called said that the patient had "blood poisoning." He gave the patient a prescription for a liquid application at night and told him to apply poultices during the day. The swelling "came to a head" on the dorsum of the toe on Monday (August 17), seven days after the infection began. The abscess ruptured on the same day, about an ounce of pus escaping The opening healed up in seven days more. He had no pain thereafter, and felt well in every way.

On the fourteenth day after the infection began (Monday, August 24) he got up and around on crutches, since he was afraid to put weight on the recently infected toe. The next day, while walking with the crutches, he slipped and fell to the ground. The weight of the body was carried to the left side. After the fall the patient found himself still holding the right crutch, which had remained in position under the right arm, his right hand on the handle. He noticed that on falling he had twisted his right forearm (internal rotation). There was no pain or snap noticed, as if he had fractured a bone. He arose, picked up his crutches, and walked six blocks further, without noticing any pain anywhere. The next day (Tuesday, August 25) his right forearm became swollen just above the wrist. He tried to use his crutches again, but could not, because of the pain in the right wrist.

The wrist gradually caused the patient more and more pain and annoyance On Saturday (August 29) he consulted his doctor, who gave him the same prescription for the wrist as previously for the toe. The patient had no definite chill at any time, but felt somewhat chilly during the night following his fall. His temperature (Saturday, August 29) was 102.5° F., and continued elevated above the normal for four or five days.

An *x*-ray plate taken on Monday (August 31) showed a fracture of the lower end of the right radius, so the patient states. A fracture of the ulna without displacement was also suspected, but could not be definitely diagnosed from the plate. A double splint was applied, one piece anteriorly and one posteriorly. The arm then felt fairly comfortable, with only a little pain occasionally. Motion in the wrist and fingers was possible and not especially painful after the splint was applied!

The patient gives no history of any previous injury to his right forearm. He has always had normal strength in the right arm. He never noticed any pain in it previous to the injury just described. His white count on September 23, 1914, the day after admission to the hospital (four weeks after his fall), was 24,000.

COMMENTS AND OPERATION

Dr. Murphy (September 23, 1914): This is a very interesting case. We aspirated this tumor of the radius yesterday, but obtained only blood in the syringe. We are not satisfied with this finding, but are going to open up the swelling to-day and explore it.

Here is the *x*-ray plate which we made of this wrist yesterday. Some bone destruction evidently has taken place in that part of the bone known as the metaphysis, as Kocher first named it. The question for us to decide is whether this lesion is an osteomyelitis or a medullary sarcoma. The history speaks strongly in favor of osteomyelitis, as does also the *x*-ray plate, and yet one occasionally sees a subperiosteal deposit of new bone, such as we have here, in a medullary sarcoma, although ossifying periostitis is one of the most characteristic and constant features of osteomyelitis. Such occasional deposits are limited to the neighbor-

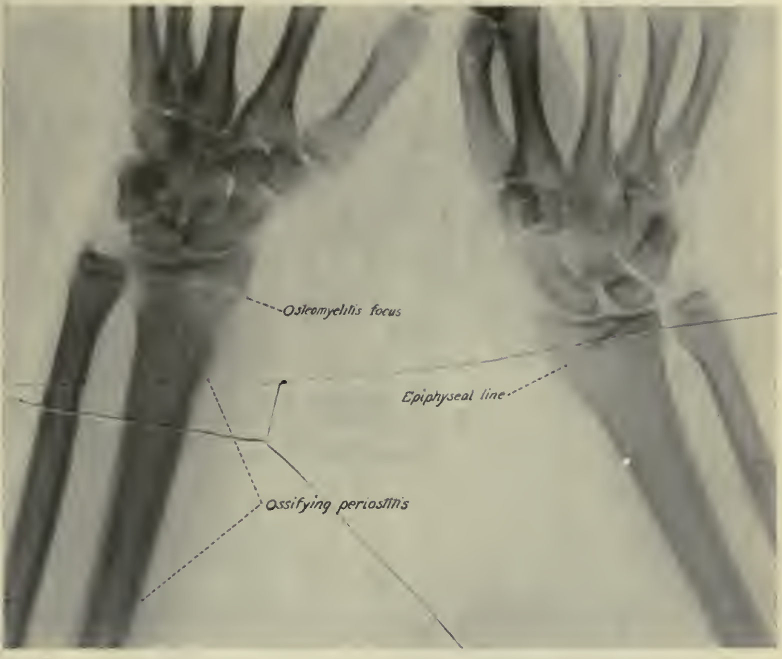


Fig. 56.—Acute osteomyelitis of the distal metaphysis of the right radius. That the focus is several weeks old is evident from the fact that bone destruction is already visible, as well as an extensive ossifying periostitis along the shaft of the radius. That the infection is not more than a few weeks in duration may be told from the fact that no material increase in the width of the bone has yet occurred and no sequestrum or involucrum formation. An acute osteomyelitis must be of at least three weeks' duration before the x-ray plate shows any evidence of its presence. The first sign is usually the development of an ossifying periostitis. The absorption of bone about the focus of infection becomes apparent only at a still later date. Therefore, the x-ray plate is of relatively slight, or, rather, of practically no, value in the early diagnosis of acute osteomyelitis.

Posteroanterior roentgenogram of both wrists made one week before operation. Note that the osteomyelitic focus has burrowed up to the epiphyseal line and already lies very close to the wrist-joint. The normal left wrist was included in the x-ray plate for the purpose of comparison.

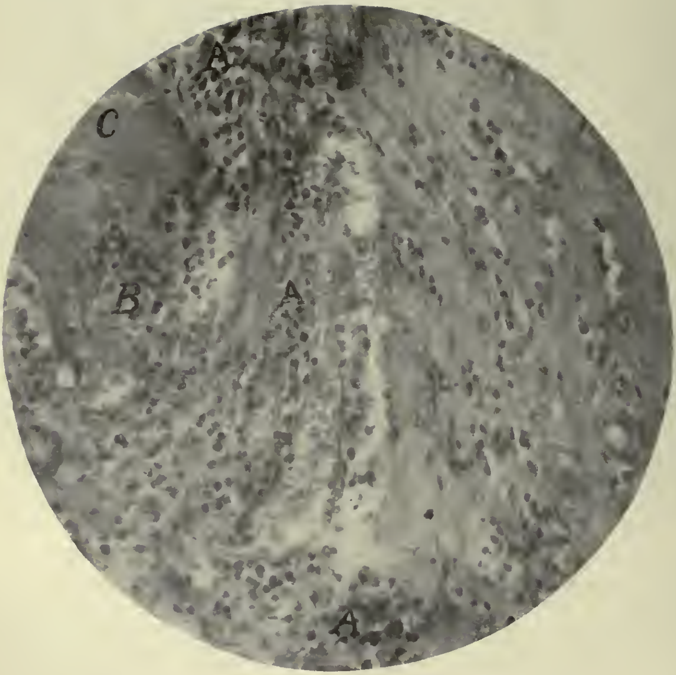


Fig. 57.—Acute osteomyelitis of the distal metaphysis of the right radius. Note the leukocytic invasion of the bone (*A*) and the lacunar absorption (*B*) going on, with osteoclasts lying in the interstices of the eroded bone (*C*).

hood of the tumor, however, and do not involve nearly or quite the entire periosteum of the shaft, as osteomyelitis so frequently does, and as is the case here The mechanism here appears to have been as follows: First an active infection of the foot, and, in consequence, pyogenic organisms circulating in the patient's blood. Then come the localizing trauma, the fall with the crutch, injuring his radius at the point where a Colles' fracture usually occurs. This accident was followed not immediately, but within the first twenty-four hours, with pain, elevation of temperature, redness, and increasing local sensitiveness to pressure, and the *x*-ray showed, as the patient came to us, a fracture of the lower end of the radius, either traumatic or pathologic in origin.

[Dr. Murphy makes a longitudinal incision over the outer surface of the swelling; that is, over the lower end of the radius, and exposes a mass of soft, friable tissue.]

Dr. Murphy: I think this is an infection in all probability; but it is still just barely possible that it is a sarcoma; so there is nothing left for me to do but to open the swelling widely. Here is a mass of granulation tissue; at least it looks much more like granulation tissue than like a sarcoma. We are sending a specimen of it to the pathologist. We are being careful not to injure the extensor tendon of the patient's thumb, and, therefore, are throwing it entirely out of the field of operation.

Now we have come to the active focus of inflammation. Here is pus. That tells the story, and clears up the case definitely. Here is the opening leading from the marrow cavity to the outside of the bone. The patient's white count was 24,000 early this morning, but that fact alone could not positively exclude the presence of a sarcoma. The opening from the medulla of the bone through the cortex lies just where one sees it in the *x*-ray plate.

I am enlarging the cortical opening so as to clean out the medullary cavity in this vicinity. The medullary focus lies just above the epiphyseal line. This looks like a low grade of infection, a not very virulent variety, because of the large amount of granulation tissue and small amount of pus. It does not look like a sarcoma at all. The bone proximal to this area of infection shows an infiltration of the medulla (just as indicated in the x-ray plate by the loss of the normal bone markings). The periosteum has developed an ossifying periostitis over the lower three or four inches of the shaft, and there is considerable edema of the soft parts just over the periosteum.

Let the record show that there was pus in the central cavity of the radius, but that there was an unusually large amount of granulation tissue in proportion to the small quantity of pus.

We are endeavoring to avoid entering the wrist-joint and to avoid interfering with the patient's radial epiphysis. We are working entirely in the bone, just above the epiphyseal line, in that portion which is known as the metaphysis. We are a little afraid of the possibility of an epiphyseal separation here on account of the patient's age, and we are doing everything possible to avoid increasing the damage.

The history of the development of this osteomyelitic infection is thoroughly in accord with our knowledge of the method of formation of infectious metastases in association with trauma. We know that if one injects infectious microörganisms into the ear vein of a rabbit and then fractures the rabbit's leg, a metastatic infection will develop in the bone at the site of the fracture in a large percentage of the cases. If one merely contuses a limb in such an infected animal, even without fracturing it, an osteomyelitis will often develop in the bone at the site of the injury. In other words, when microörganisms are circulating in the blood-stream trauma permits their escape from the blood into the injured tissues, and furnishes a *locus minoris resistentiæ* for their growth.

That is exactly what happened to this patient. The infectious microörganisms were circulating in his blood and the trauma to his right wrist caused by his fall produced a site favorable to the development of an osteomyelitis of the typical variety in the patient's radial metaphysis.

The condition is not a tuberculosis. Tuberculosis can be ruled out practically from the clinical history alone. Why? Because the present lesion has developed too rapidly for a tuberculosis, the patient's hand being incapacitated almost immediately after the injury. He had a fall, and the evidences of an acute osteomyelitis developed within a few hours. That history speaks also against the lesion being a sarcoma, because the patient was incapacitated so immediately after the injury. It is only the round-cell, aneurysmal variety of sarcoma which has so rapid a destructive action on bone as an acute osteomyelitis. With the round-cell aneurysmal sarcoma the patient's affected limb may become incapacitated for use inside of forty-eight hours after the initial trauma. I have had just such a case. The occurrence is very rare, however. With such a sarcoma there may be a leukocytosis and fever, and the condition may look exactly like an infection until one incises it, and the blood from the arterial sinuses shoots all over the field of operation.

We are sterilizing the focus in the bone with 95 per cent. carbolic acid, and one minute later neutralizing it with alcohol. We are going to close the wound completely and try to obtain a primary union and a Schede blood-clot to fill the cavity.

Pathologist's Report (Dr. W. O. Sweek).—The soft fragments removed at operation consisted only of granulation tissue. The pieces of bone excised showed infiltration with round-cells and leukocytes, together with areas of lacunar absorption and new vessel formation. There is nothing in any of the specimens suggestive of sarcoma. Only the usual histologic appearances of a mild acute osteomyelitic focus are present.

[NOTE.—The wound opened again in four or five days and discharged a serosanguinolent fluid. The osteomyelitis then extended slowly up the radius. Some weeks later the radius was again opened up, the central cavity cureted and packed with gauze to produce healing with granulation from the bottom. The defect is still slowly filling in.—ED.]

BONY LIPPING OF THE RIGHT ACETABULAR MARGIN AND OF THE NECK OF THE FEMUR FOLLOWING A METASTATIC ARTHRITIS.—ARTHROPLASTY OF THE HIP.—CHEILOTOMY

HISTORY

The patient is a married woman, aged twenty-eight years. Her family history is negative as to tuberculosis, carcinoma, and rheumatism. She had pneumonia when sixteen years of age, and was ill for six weeks, with no complications. She has always been subject to colds and sore throat. She has been married for eight years and has one child. Her menstrual history is negative.

Her present trouble dates back to seven and one-half years ago (1907), when she was two months pregnant. About 11 A. M. one day she noticed a severe pain, cutting in character, in the calf of the right leg. There was no swelling or redness visible, and she had no chills or fever to her knowledge; but the pain became so severe that she went to bed and remained there for three months. When the knee was elevated the pain was somewhat relieved. She had no pain in the lumbar region. No injury preceded the onset of the pain. About the same time that her right leg was troubling her she noticed some numbness in the toes of the left foot.

At the end of three months she was up and around with the aid of two canes. The pain was still present in the right leg, and there was also some shortening of the limb; later a slight curvature of the spine developed She was unable for a long time to move her hip much on account of the pain caused by motion. Her pregnancy went on to term and she gave birth to a normal child after a normal delivery. After delivery the condition in the hip continued the same. The motion in the joint seemed limited and there was pain on both motion and pressure.

One year after the onset of the trouble she was given an anesthetic and the joint was forcibly manipulated. A cast was then put on for nine weeks without abduction of the leg! This did not improve her condition, which remained practically unchanged during the following year. She then took treatments from an osteopath for two years. After these treatments her pain was somewhat relieved, but the partial ankylosis of the hip still exists and was not influenced.

COMMENTS AND OPERATION

Dr. Murphy (November 21, 1914): The patient has practically no abduction left. She has a little adduction remaining [indicating], but no abduction. Let us go over the clinical course of this case and pick out the salient points in it. First, there appeared one morning a sharp, sudden pain, which the patient states was confined to the calf of the right leg, but with no sensitiveness to pressure. The next day the pain appeared in her right hip and it became so severe that she would not permit motion in the joint. She was, thereafter, in bed for three months, always with intense pain in the hip. The history states that she had no temperature, so far as she can remember. I do not recall the patient's statement to me concerning fever. If she had no temperature, she was a very great exception to the rule in this class of cases, because the rule is that such patients almost always have at least slight fever, and, in the early days of the disease, during the onset, they have a high fever. I doubt the statement that she had no rise of temperature. It is more likely that during the seven years which have intervened she has forgotten the details of the onset. The cases of metastatic coxitis with a mild onset are very prone to develop periarticular osteophytes, first around the margin of the acetabulum and then on the neck of the femur. Subsequently they may develop a fibrous ankylosis or a bony ankylosis. But the cases which are initiated with a chill and high fever practically always develop a bony ankylosis in about three or three and one-half months after the onset.

The osteophytes in the mild cases are particularly prone

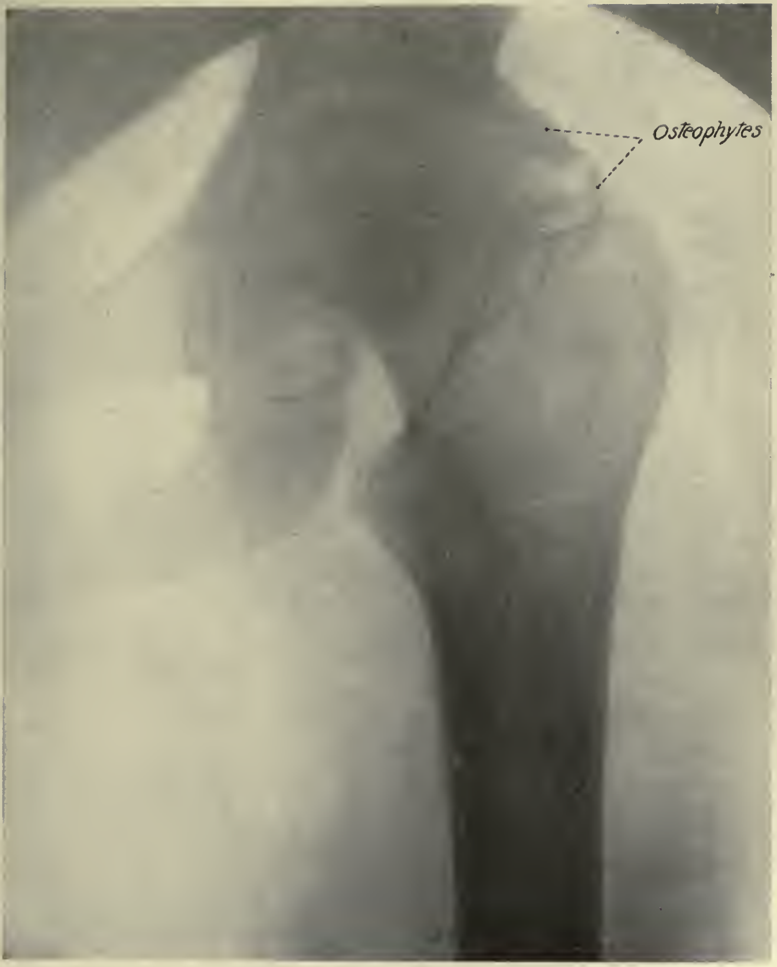


Fig. 58.—Bony lipping of the margin of the right acetabulum and of the neck of the femur, following a metastatic arthritis. Anteroposterior roentgenogram made shortly before operation. Note how much more marked is the development of osteophytes at the upper margin of the acetabulum and on the upper surface of the neck of the femur, than on the respective lower margin and lower surface. The consequent diminution of abduction is obvious.

to develop on those portions of the acetabular rim and on the neck of the femur, where they limit or completely inhibit abduction. The present is a case in point. The deposition of bone around the margin of the acetabulum and around the neck of the femur can be easily outlined here in the *x*-ray plate. The "lipping" is most marked and most easily seen at the outer upper margin of the acetabulum and on the upper surface of the femoral neck. The bone deposits at the lower and inner margin of the acetabulum and on the lower surface of the femur are minimal and of minor importance. In these mild cases the great deposit of bone occurs regularly in this position on the outer and upper edge of the acetabulum and the upper surface of the femoral neck. In the very similar case operated last week the deposit on the upper surface of the neck came well up on the external margin of the head, practically eliminating all power of abduction. In this case also the upper acetabular and femoral osteophytes impinge on each other, so that abduction is completely impossible, although about four inches of adduction are still present. The limb lies in a position of permanent adduction. She has not a bony ankylosis of the femur to the ilium. If she had, she would be free from pain, which she is not, because pain has been and is one of her chief complaints. The rubbing of these osteophytes on one another produces a constant irritation, and the irritation increases the ossification in the fibrous layer of the capsule. Remember that the fibrous layer of the capsule is the continuation of the fibrous layer of the periosteum, and it is in this fibrous layer that a marked tendency to ossification exists. Thus these cases of partial ankylosis tend to develop a complete ankylosis. In the embryo hip there is no joint at first—only a single mass of cartilage. Then the cartilage splits transversely and then a synovial membrane develops at the split. The separation of the two masses of cartilage becomes complete to the margin of the joint. Then comes the detachment of a portion of what was originally the periosteum or the perichondrium, to form a capsule for the joint.

Whether we shall do a complete arthroplasty or whether we shall perform only a lipping operation, a removal of the bony prominences, depends on what pathologic condition we find when we get into the joint. We prefer to do only the lipping operation when it promises a reasonable probability of cure. The lipping operation, cheilectomy, results in great relief to the patient, when technically successful, for the patient is made free from pain. The technic of the procedure is practically the same as the technic of our hip arthroplasty. We chip away the osteophytes, but omit reaming out the acetabulum and milling the femoral head.

Even when we are examining her under the anesthetic, as you note, there is not much motion and there is *no* abduction. If the condition was allowed to run its natural course for a few more months or years, a complete ankylosis would result. I am marking out the field for my incision.

[Following his usual technic, which was described in full in the August, 1914, number of the Clinics, Dr. Murphy exposed the joint.]

Note how the joint capsule has become thickened over the femoral osteophytes. Now we have exposed the lipping of the acetabular margin. Note how elongated the bony projections are at the upper acetabular margin, just as the *x*-ray plate showed them. (See illustrations, Figs. 59 and 60.) Note the line of demarcation between the joint cartilage and the bony lipping of the acetabular margin.

Next we must divide this "Y" ligament, which will otherwise be a source of great trouble to the patient subsequently when she begins abduction, because of its contracted state. Note how far forward the mass of osteophytes lies on the neck of the femur, encroaching even on the articular surface of the head. Now one can see very beautifully how the bony mass on the neck impinges against the lipping osteophytes of the upper acetabular margin and prevents abduction. The depth of the true acetabular cavity appears to be normal, and the joint surface seems practically free from adhesions. The marked changes here have lain at the margin of the joint rather than on the articular surface.

In removing these osteophytes we must make certain to remove the covering periosteum with them; otherwise they are likely to recur. This acetabular margin which we are removing looks like a regular collar of bone, but it is very much thicker

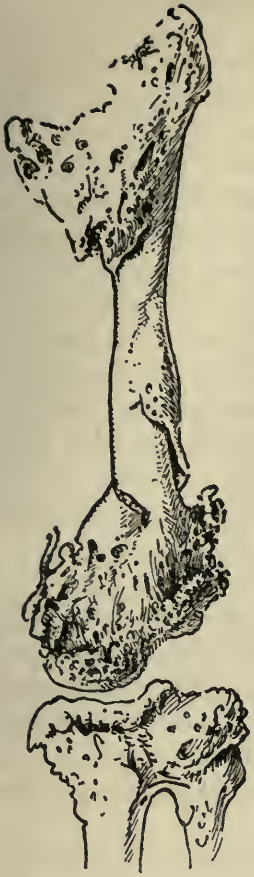


Fig. 59.—Enormous "lipping" of the articular surfaces of the knee-joint in a case of chronic arthritis of great severity—a Charcot's joint. The patient was a tabetic. This maximum grade of lipping is not very often seen. In the lower extremities it occurs almost exclusively with tabes, and in the upper extremities with syringomyelia. But the slight to moderate grades are very common and quite constant in all cases of chronic arthritis, in the diagnosis of which the demonstration of "lipping" is a matter of prime importance, since it is pathognomonic and, probably, the earliest objective sign to appear. (Specimen from Dr. Murphy's bone collection.)

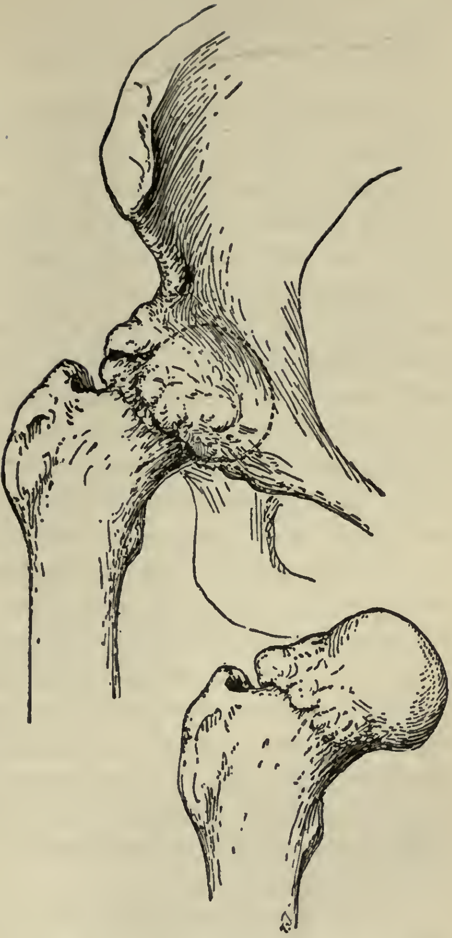


Fig. 60.—Bony "lipping" of the acetabular margin and of the neck of the femur following a metastatic arthritis. Note how the interlocking osteophytes completely prevent abduction although interfering only moderately with adduction. The joint cavity itself was relatively free from adhesions, the pathologic changes being almost entirely confined to the articular margins. Therefore, the interarticular interposition of the usual pedicled flap of fascia and fat was not necessary in this case, because the joint movements became free as soon as the ankylosing osteophytes were chiseled away.

above than below, as I have previously remarked is the rule, and as the *x*-ray showed us. [Excises all the osteophytes.]

How easily and completely we can abduct the patient's leg now the acetabular margin has been removed, together with the bony mass on the upper surface of the neck of the femur which impinged against the former! That result verifies our diagnosis and ought to give the patient a very satisfactory joint. In this case we shall not need to interpose a flap of fascia and fat in the joint, since the articular cavity is in such good condition. We shall use our pedicled flap, however, but shall place it around the neck of the femur to prevent fresh adhesions forming around the neck. This is the same pedicled fascia-and-fat flap that I would have used had I made the interarticular interposition as usual. I am also covering the raw bone surface on the acetabular margin with a pad of fat to prevent adhesions forming to it, and to diminish the liability to new bone formation.

This is the most common pathologic condition that one finds in the hip-joint in these metastatic coxitis cases. This partial ankylosis due to "lipping" is much more common than complete bony ankylosis, and is more serious for the patient, in that he enjoys practically no more motion than with a complete bony ankylosis, but has nearly always more or less pain in the joint, especially on motion.

Formerly we nailed the sawed-off trochanter back into place, but we experienced some trouble with the nails, so now we do not nail the trochanter, but suture it back into position with phosphor bronze wire, 16 strands woven together, or chromicized catgut. This wire heals in very readily. Even if the wound suppurates, it will usually heal in, even if a hematoma develops.

Let the record show that we also performed a subcutaneous tenotomy on the contracted adductor tendons in order to insure perfect freedom of abduction.

The patient will be placed, as usual, in a well-padded Travois splint, both legs being widely abducted.

[NOTE.—January 16, 1915: The wound healed by complete primary union. The patient has a good range of motion and excellent abduction. She left the hospital to-day for her home in New York, and *she walked out!*—ED.]

CARCINOMA OF THE BREAST

Dr. Murphy: We are very fortunate in having with us to-day one of the world's great teachers and surgeons, a man who has been honored by the American Medical Association and who has honored the American Medical Association by becoming its president. I have requested him, and he has very generously consented, to give us a talk to-day on the subject in which his name is a household word, and with which every doctor is familiar. I feel that an hour or two of teaching from him would place his attitude on the cancer problem clearly and forcibly before us. I have the pleasure of presenting to you Dr. William L. Rodman, of Philadelphia, president of the American Medical Association, who will take up the subject.

Dr. Rodman: I am afraid, gentlemen, that you will be very much disappointed after the very generous and flattering introduction by Dr. Murphy. Nevertheless, I esteem it a great privilege to speak at a clinic that represents in every way all that is best in American surgery. I also appreciate the opportunity and privilege of speaking to so many surgeons from different parts of the world, and demonstrating a method of radical amputation of the breast which has served me well for the past seven years. The method also has been used by others who have thought sufficiently well of it to recommend it to their friends.

As Dr. Murphy has requested, I shall take up the phase which he thinks, and which every one believes at this time, is the most important one pertaining to the cancer question—precancerous conditions, or at least earlier diagnoses than have been hitherto practised. If we, in other words, are going to operate only on cases of cancer of the breast which can be diagnosed clinically, then we shall never get very much better results than were secured fifteen or twenty years ago. It is, however, by making the diagnosis earlier and then applying the treatment at once that better results are being obtained. It would be very interesting, and I wish that I had the time to do it, to take up the history of operations for cancer of the breast and to show how they have improved decade by decade. If every year has not brought an improvement in statistics, each quinquennium has done so. We all know that when S. W. Gross was preaching radical operation, back in the late seventies and early eighties,—and he was rather radical for his time,—he was looked upon as a misguided optimist, because he claimed to cure 9 per cent. of his patients with cancer of the breast. He was the only one making such a claim in this country. His distinguished father, Agnew, his contemporary in Philadelphia, and Sands, of New York, said they had amputated "cart-loads" of breasts and had never seen a radical cure. Then, five years later, Gross's results had so much improved that in his last publication (1888) he reported 21.5 per cent. of cures. Later on Bull reported 26+ per cent. of three-year cures and Halsted even better results (44 per cent.), and these statistics were based upon operations performed on patients in whom a clinical diagnosis of cancer was possible.

There are a few essential and basic facts that one ought to carry in one's mind in regard to neoplasms of the mammary gland. Certainly benign tumors are more common than was formerly believed. Gross, in his classic monograph on the subject, said that only about 9½ per cent. of the tumors of the mammary gland were benign. An analysis of a large number of cases—more than 5000—taken from the German clinics convinced me that 16½ per cent. of the tumors of the mammary gland are benign. If we add to this the 2½ per cent. of sarcomata, you see that we have practically 19 per cent. of non-carcinomatous growths of the mammary gland. There is another condition that was little thought of in Gross's day, and which is more common than benign neoplasms of the breast, viz., chronic cystic mastitis, the disease of Reclus, or, as the Germans call it, Schimmelbusch's disease, or, as I like to call it, *abnormal involution* (Warren). The definition of Dr. Whitney, of Boston, if you want a pathologic definition,—and it is, I think, more accurate,—

is fibrous and glandular hyperplasia with retention cysts. That term tells one accurately the pathology of such breasts. The hyperplasia is the initial stage, and as a result of pressure, cysts follow. Now, Reclus, although he wrote so well and convincingly on this subject in 1887, was not given the attention that should have been given to him at that time, as he called this disease *intra-acinous cystic epithelioma.* If his teaching had been fully appreciated at that time, and the disease had taken its pathologic name, rather than the name of the one who described it, how much better it would have been, because he definitely recognized what others did not recognize for many years, that it is frequently the forerunner of cancer! Latterly a great deal of attention has been given to this subject. Dr. Murphy, in the last publication of his CLINICS, quoted Greenough and Simmons and other workers in this field. Greenough and Simmons found in their series that 10 per cent. of their patients showed carcinomatous degeneration; Dr. Warren, of Boston, encountered 13 per cent. undergoing malignancy, and Speese, of Philadelphia, reviewing all the cases in the University Hospital, found that 25 per cent. of them were undoubtedly carcinomatous. A recent German publication (Bertel) states that exactly 25 per cent. of abnormal involution cases became carcinomatous. In a recent and careful analysis of 65 cases in my own practice, I found that 14, or 21.5 per cent., had undoubtedly become carcinomatous at the time of operation. In other words, the process in all the 65 cases began as a benign one, but in some had changed into a carcinomatous one at the time of operation. This, I think, is very important to bear in mind.

This is the mammary condition most difficult to distinguish from carcinoma. The differentiation can be safely made only with the microscope. It is a comparatively easy matter to recognize the clinical difference between a solid benign neoplasm and a cancer of the breast. It is more difficult to differentiate between benign and malignant cysts, and, as I have said, next to impossible to discriminate between cancer and advanced cases of chronic cystic mastitis. To add to the difficulty, both are more likely to occur between forty and fifty, as in women near the menopause.

Still, there are essential, if not pathognomonic, differences between the two diseases which may at times be so accentuated as to make a differential diagnosis easy. In the first place, abnormal involution is frequently bilateral; cancer, very rarely so. Recently I saw and operated upon the second case of bilateral carcinoma I have ever seen in my life. She was a patient in whom the lesion seemed to appear in each breast at the same time. I have seen many times, of course, secondary tumors in the opposite breast. The most important difference between the two is that abnormal involution is painful and cancer is not. Cancer rarely gives any pain during the first year of its existence. I wish to impress upon you that fact and to emphasize it as strongly as possible, because nearly all the text-books state that cancer is a painful disease. Of course, it is in the later stages, when ulceration occurs and when adhesions to the skin and to the costal wall beneath take place; but in the early stages of cancer it is absolutely painless, differing entirely from an inflammatory condition. Moreover, the pain in chronic cystic mastitis is variable, nearly always being increased just about the menstrual period. During that time also the breast enlarges visibly, to subside when the period is over. Pain is markedly increased when pressure is made over certain areas more sensitive than others. This will usually be over sites beneath which cysts are found, or an area where the inflammatory changes are most marked. That picture shows very well a typical chronic mastitis with small cysts scattered throughout the breast. Enlarged veins are frequently seen in the skin superlying the affected breast. I do not know that others have called attention to it, but I have observed it so often that I always look for it. The veins are not as much enlarged as often found in sarcoma, but are visibly so. That is a good point, and something that you will not find in cancer of the breast. Further, there is never adhesion of the skin to the breast in abnormal involution. Such adhesion is usually found even in the early stages of cancer. Glandular involvement more frequently takes place in cancer, but it does occur in abnormal involution. The difference, however, is that the axillary glands, which are enlarged in abnormal involution,

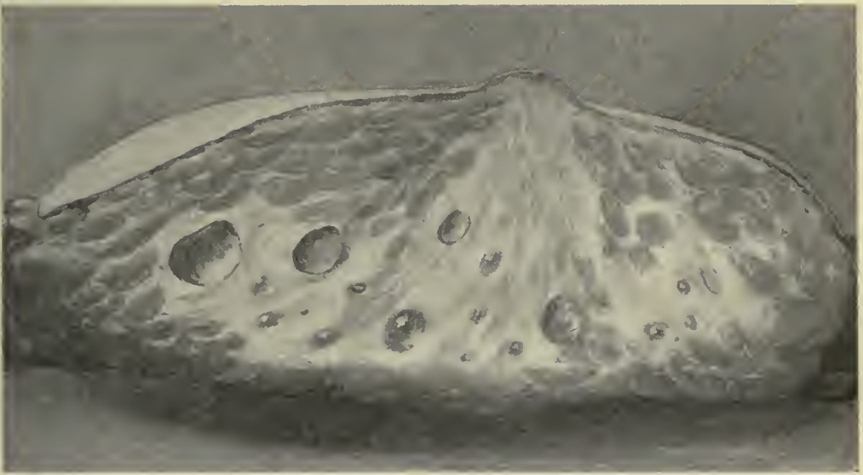


Fig. 61.—Abnormal involution of the breast (general cystic disease). Gross section of the breast passing through the nipple. Note the cysts of varying size scattered through the mass of connective tissue (Warren).

are smaller, and, being the result of inflammation, are painful—very much so upon pressure. The enlarged glands in cancer are not. The enlarged glands may coalesce, become matted together, and form one large mass when cancerous, which is not the case in abnormal involution. Not infrequently there will be more or less discharge from the nipple in abnormal involution. That, of course, is very infrequent in cancer. In abnormal involution there is oftentimes a clear, watery, or straw-colored serum exuding from the nipple, or made to do so upon pressure, if the adjacent portion of the breast be diseased. In cancer the discharge is sanious. If pure blood, it is significant of another benign condition—papillary cystadenoma, which may be an advanced stage of abnormal involution. I agree again with Dr. Murphy, who stated, in his recent article, that these papillary cystadenomata, while they are properly classed as benign originally, are nearly always eventually malignant. Five out of six of my cases were definitely malignant, hence I treat every case of papillary cystadenoma as malignant, doing a complete operation, just as I would in a well-marked case of carcinoma. Now in about 25 per cent. of benign tumors, and in a larger per cent. of abnormal involution cases, the most experienced clinician will not be able clinically to differentiate them from cancer. The only safe procedure in such circumstances is to have present a competent pathologist, prepared to make frozen sections at once from the most suspicious tissues submitted to him. His report comes back in five or six minutes, and you will be governed accordingly. If it proves to be a simple case of abnormal involution, one which has not undergone malignant change, then you may remove the breast, retaining the pectoralis major muscle. If malignant, you will, of course, remove both muscles in all instances. If you are in doubt, or the microscopist is in doubt, then I insist, in all instances, upon a complete operation, because we find that the functional use of the arm is not in the least impaired after a radical operation, and it is very discouraging, humiliating, and embarrassing to find that the report from the frozen sections is not confirmed by the serial sections which are made later, and are, of course, more accurate. With competent pathologists, they will very rarely disagree. It has happened with me only twice, and one of the pathologists had never undertaken the use of frozen sections before, and only served in the absence of my regular pathologist. Remember that any pathologist examining the most diseased part of the breast will give you just as accurate a report on frozen sections as he can give you after making serial sections later on; but *you* may fail to get the most diseased part of the breast, and, therefore, his report may mislead you. So I insist that in cases of doubt it is much better to do a complete operation in abnormal involution, because, at the very least, 20 per cent. of these cases are malignant at the time of operation, and that in itself is sufficient justification for doing a radical operation in the majority of instances of advanced Reclus' disease.

Dr. Greenough has reported a number of cases treated by the operation called "plastic resection." The breast was lifted up by the Warren method, and a plastic resection or partial removal of the gland practised. Later reports showed a number of recurrences that took place after partial removal of the mammary gland for abnormal involution, and not an inconsiderable number were not only markedly malignant, but from an operative standpoint practically hopeless. When one fails to remove every particle of a malignant focus, the chance for cure has practically been greatly lessened, but I will not say lost. I know that I have saved two patients by second operations, as both have now passed the five-year limit. One has gone nine and one-half years. But "ever" and "never" are words which I think should not be used in medicine and surgery. There are always exceptions. If one has a patient twenty years of age with abnormal involution, and it seems to be limited, then, as her whole future and hope of happiness lie before her, one is justified in taking a chance and not doing a complete or radical operation; but such patients should always be given the facts, should accept the responsibility themselves, and should be made to appreciate the necessity of keeping in close touch with the surgeon, so that if there should be the slightest evidence of future trouble, a radical operation could perhaps be done immediately. I insist, however, that these cases are exceptional, and that the rule should be a

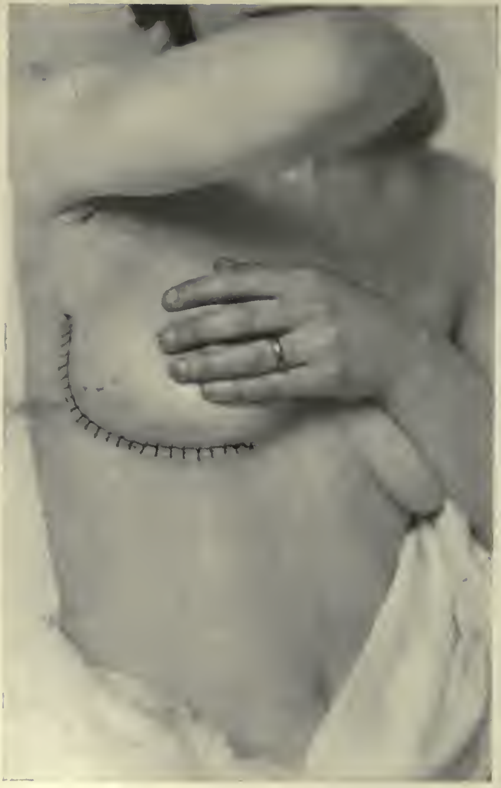


Fig. 62.—Dr. Rodman's incision for plastic resection for benign tumors of breast.

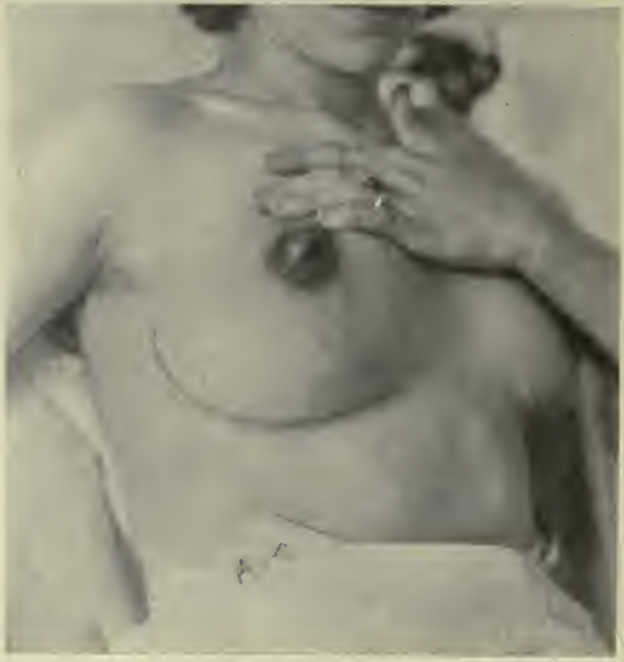


Fig. 63.—There is little or no deformity after plastic resection, as the scar is hidden by overhanging breast.

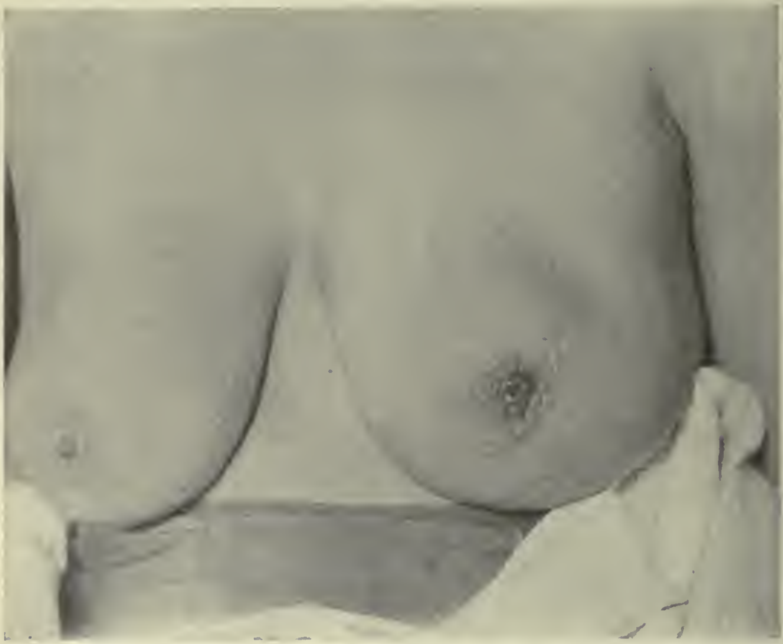


Fig. 64.—Paget's disease. Left breast.

complete removal of the breast, even as one would do in cancer.

There is another so-called precancerous condition, viz., Paget's disease, of which I should like to speak. I was very much pleased with Dr. Murphy's recent report on this disease, accompanied by a beautiful colored drawing depicting a typical case (see August number of Clinics). I have rarely seen so good an illustration of the disease. He takes the same view that I have always held of Paget's disease, that it is not primarily a disease of the skin advancing inward. The cutaneous involvement is secondary, and is caused by the discharge which comes from the diseased tissue within. It is duct cancer. Not only is Paget's disease, so far as my experience goes, cancerous *ab initio*, but it is one of the most fatal forms of cancer that I encounter. Nearly all the patients with it that I have seen have promptly suffered recurrences after operation. I consider most of the teaching on the subject absolutely wrong. The accompanying photograph illustrates my point. The excoriation of the nipple and areola had only appeared fourteen days before the host reported for operation. It was performed the following morning. The entire breast was badly involved, and all the axillary glands infected up to the clavicle. The supraclavicular glands were explored, but apparently were not diseased. In spite of frequent *x*-ray treatments, she now has, eighteen months after operation, supraclavicular metastases.

Papillary cystadenoma, which is only an advanced stage of abnormal involution, can usually be differentiated from cancer in a clinical way, because this trouble is nearly always immediately behind the nipple. The discharge of pure blood, the central location of the cyst, the age of the patient, usually about forty-eight or forty-nine years, on an average, will enable one to recognize it. But, as I have said, it matters little, for from its inception it is potential cancer and demands radical operation. I have found but one exception. Of the six papillary cystadenomata that I operated on and have records of five were definitely malignant; one was not.

We often hear that cancer of the breast is much more common than any other mammary disease. In an analysis of 200 consecutive cases in my own private practice I found:

Carcinomata	83	(41.5 per cent.)
Abnormal involution	67	(33.5 per cent.)
Benign tumors	30	(15 per cent.)
Papillary cystadenomata	6	(3 per cent.)
Tuberculosis of the breast	6	(3 per cent.)
Sarcoma	5	(2.5 per cent.)
Galactocele	3	(1.5 per cent.)

Gross estimated that sarcomata comprised more than 8 per cent. of the tumors of the mammary gland. We now find it a rare neoplasm in the breast, because refinements in pathology have made us understand that what were formerly called sarcomata, are in reality myxomata or other growths. Its rarity in the breast is in striking contrast to its frequency in other parts of the body. Statistics from the Johns Hopkins Hospital indicate that it is even more rare than we have found it, comprising only 1.5 per cent. of mammary growths.

Now we come to the question of the technic of the operation for cancer of the breast. We might say that the complete or radical operation, as it is understood in America, indeed, everywhere, was introduced to the profession by Halsted and Willy Meyer in 1894. In other words, they were the first to advise it routinely—not the first to practise it in individual cases. Volkmann, Billroth, and Gross had all removed the muscles years before, but Halsted and Meyer advised the removal of the muscles in every case, thereby facilitating a complete axillary dissection, for, be it remembered that *the muscles are removed not because they are involved in the carcinomatous process*, as so many think, *but simply for the reason that a thorough axillary dissection cannot be made with the muscles in situ.* One may think differently, but it cannot be done. In the technic which has been so generally followed by nearly all surgeons, the work is done from the sternum to the axilla, and undoubtedly most excellent results can be secured by following the Halsted method. Halsted himself secured 44 per cent. of cures; others have done equally well. It seems to me, however, that it is much better to work from the axilla to the sternum. The reasons are surgical—they are anatomic—they are pathologic. In the first place, one can the better plan his incision so as to keep away from the arm and not interfere with its future usefulness. In the second place, one can ligate the vessels at their roots and practically do away with hemorrhage. It is surprising what a difference it makes to ligate the vessels at their roots, rather than to begin at the sternum and cut and recut time and again the same vessels and have to tie them repeatedly. More important than either of these reasons is that one works from healthy toward diseased tissue. One of the greatest possible dangers to the patient is the expression of cancer-cells during the operation, forcing them into the lymphatic vessels and thence into tissues more or less distant from the primary focus. Now this you avoid by blocking the lymphatics in the axilla at once. Another and a very good reason for beginning in this way is that you may find the axilla so extensively involved that a radical operation will be futile, and it may be unwise to subject the patient to the risk of a complete operation. Now if that be so, the sooner you know it the better, rather than discover it after the operation has been practically done and the breast removed. These reasons seem to me good ones for reversing the technic and working from the axilla to the sternum, rather than conversely.

Now if the doctor will be kind enough to show the slides, I think we will get a better idea of the technic than we can get from words.

First picture: The first incision begins 1 inch below the clavicle, and two fingerbreadths to the inside of the arm, or internal to the sulcus, where the pectoral muscle joins the deltoid. In this way the cephalic vein will be avoided. Your incision is kept away from the arm, and the cicatrix which results never in any way encroaches upon the vessels or interferes with the movements of the arm. In a person of average size and stature the incision will be five inches long; in small women, four and one-half; in large women, six or more. The incision is straight, parallel with the arm, two fingerbreadths to the inside. Cut rapidly down to the pectoralis major muscle. There are no vessels of any size severed.

Second picture: Now you thrust your index-finger under the lower border of the pectoralis major muscle, and, as a rule, let it emerge between the costal and clavicular portions of the muscle. If the growth is peripherally situated and in the upper hemisphere, the entire muscle should be removed. Cutting the muscle at its insertion is facilitated by the employment of a six-prong retractor devised for the purpose, as it enables the skin and superficial fascia to be easily withdrawn outward, and the muscle followed to its insertion into the humerus.

Only a slight dissection will be necessary to discover the lower edge of the tendon of the pectoralis minor. This should be separated from the fascia covering the tendon and below it. Otherwise the long thoracic artery which runs in the fascia parallel with and just below the tendon may easily be wounded.

The index-finger is now introduced underneath the muscle and made to emerge at its upper border. Lifting up the muscle, the tendon is made tense and prominent, so that it can readily be seen that no other tissues are included with the tendon. The acromiothoracic artery runs just above and parallel with this tendon, and, being a branch of considerable size, might cause some little embarrassment if it were cut at this stage of the operation. The tendon is divided at its insertion into the coracoid process. Therefore we have the acromiothoracic artery parallel with and just above the upper border of the minor pectoral tendon; the long thoracic parallel with and just below its lower border. Both can easily be avoided if care is taken. I have never as yet wounded either vessel, nor is there excuse for doing so. Both muscles retract inward as soon as their respective tendons are severed. This at once uncovers the axilla and makes its subsequent thorough dissection easy. The costocoracoid membrane is now opened and largely sacrificed, which gives ready access to the subclavicular fat at the apex of the axilla. In removing a part of this membrane the cephalic vein at the upper and outer aspect of the wound must not be wounded. There is also in the fascia a branch of the acromiothoracic which, with its accompanying vein, should be clamped and tied. A nerve

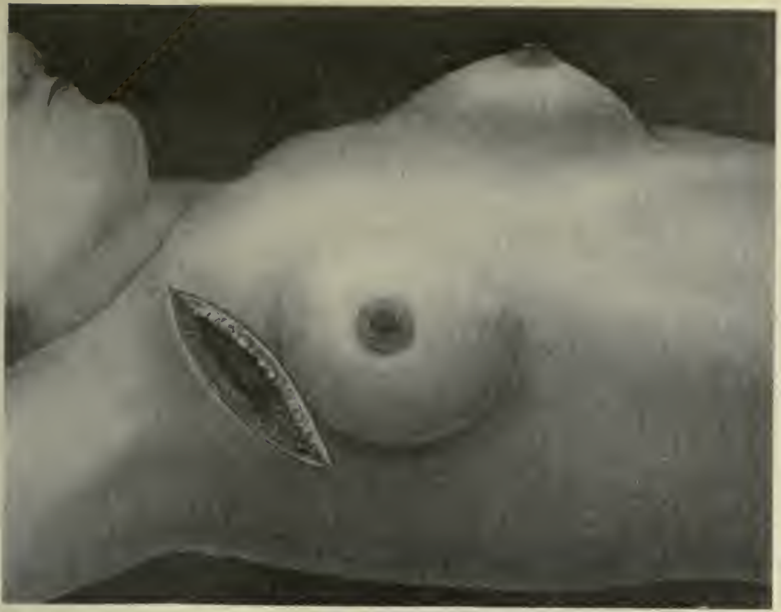


Fig. 65.—Dr. Rodman's operation for cancer of the breast. Primary skin incision (Rodman, "Diseases of the Breast").

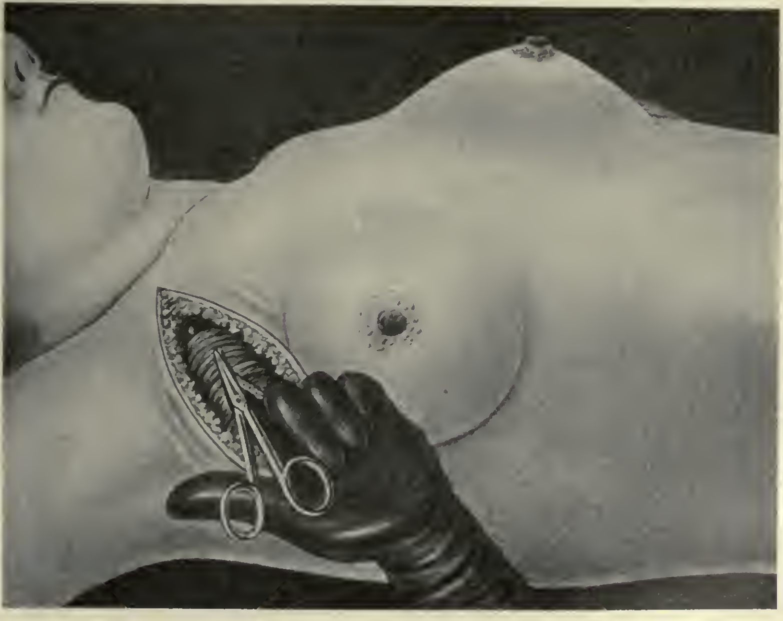


Fig. 66.—Division of pectoralis major near its insertion (Rodman, "Diseases of the Breast").

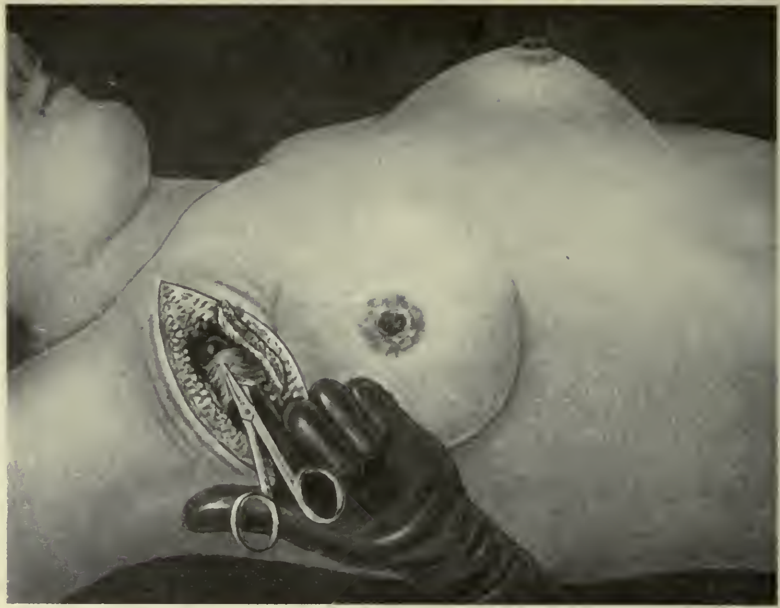


Fig. 67.—Division of pectoralis minor at its insertion (Rodman, "Diseases of the Breast").

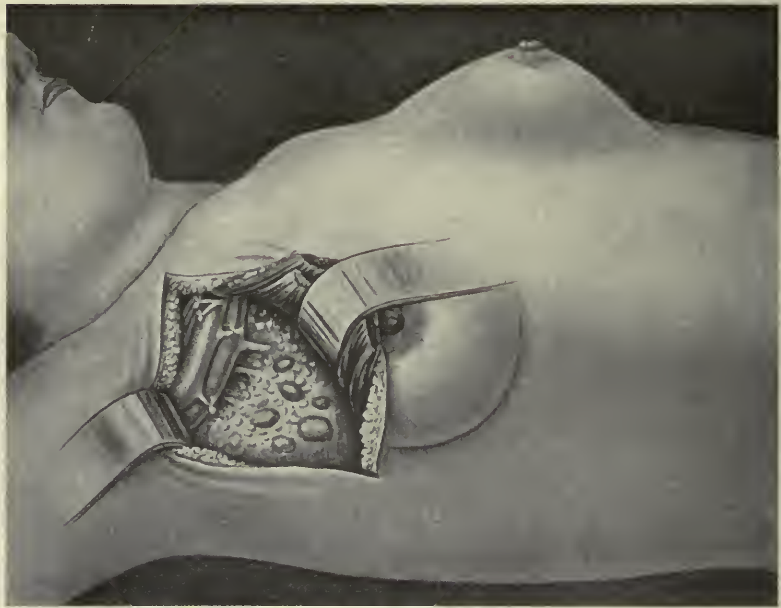


Fig. 68.—Axilla freely exposed and vessels divided at their origin. The small vessels to this area are tied and cut close to the axillary vessels (Rodman, "Diseases of the Breast").

supplying the pectoral muscle may as well be sacrificed now, as it necessarily must be later on when the muscles are removed.

The dissection is begun at the apex of the axilla and must be most carefully conducted, lest injury be done to either the axillary vein or the acromiothoracic artery. It should be from above downward, though this is perhaps somewhat more difficult than making the dissection from below upward.

In the removal of the fat and fascia in the upper third of the axilla the finger, covered by several thicknesses of gauze, will be all that is necessary. Instruments are rather dangerous unless used most cautiously; moreover, they are unnecessary.

An incision is now carefully made through the fascia to the outer side of the axillary vessels simply to start the dissection from without inward. This is made to the extent of the lower two-thirds of the axilla, but not in the upper third, where it is dangerous to cut. I continue the dissection largely with gauze, but Allis's or Mayo's blunt dissectors may be used freely and are most helpful. Occasionally a cut with scissors or a sharp knife facilitates the dissection. The instrument of Charles H. Mayo is more than a blunt dissector, for it can be used as scissors and is most valuable in economizing time, making a change of instruments unnecessary.

As the sheath and fat are removed from the vessels we come down upon the acromial, long and alar thoracic branches, and the subscapular branch of the axillary artery, in the order named, from above downward, which, with their accompanying veins, are to be carefully clamped in two places and divided between. Both ends are ligated. In this way the subsequent hemorrhage is materially lessened; in fact, it is surprising how little blood will be lost during so prolonged and extensive a surgical procedure.

The enlarged lymphatic glands will usually be found at the base of the axilla, between the latissimus dorsi, teres major, and subscapularis muscles posteriorly; the serratus magnus internally; and inferior to a line formerly indicated by the situation of the lower border of the pectoralis minor. The midaxillary and subclavian glands may, however, be infected. All such enlarged glands and surrounding fat should be carefully dissected from the several muscles, and to do this best the fascia covering the muscles must be sacrificed. In fact, so thorough should be the axillary dissection that nothing is left on its inner aspect save the posterior thoracic or nerve of Bell; on the posterior aspect the long subscapular nerve, and superiorly, possibly, the superior thoracic artery, if it arises as an independent branch high up on the first portion of the axillary. In such circumstances it is impossible, in my judgment, to reach it with safety. It is so deeply placed that there is great danger of doing serious damage to the vein and artery, the former particularly if an attempt is made to secure the vessel at its root. I have convinced myself by work in the dissecting-room that it is at least a hazardous, if not an unwarranted, procedure. It is a small branch and negligible so far as subsequent hemorrhage is concerned. Moreover, it not infrequently arises conjointly with the acromiothoracic, and in such cases is easily secured with the other vessel.

A thorough dissection of the axilla can usually be finished in fifteen minutes, and is entirely accomplished through the single straight incision. It should invariably be from above downward, without inward, and *en masse*. A piecemeal extirpation is not to be considered. Patience, a good light, and working with blunt dissectors will insure a safe and reasonably speedy dissection of the axilla. As we have said, sharp instruments are not to be used at the apex of the axilla, but may materially facilitate the dissection at its base. Accidental injuries to the vein are not common, and when occurring are, if practicable, to be treated by lateral ligature or suture. We have never accidentally injured the vein, but have deliberately resected it eight times when its walls were seemingly infected and closely adherent to enlarged and infected glands.

The axillary dissection having been finished, a second incision is made from the middle of the original one. It goes well above the breast, extends to the middle of the sternum, and then passes downward to a point midway between the ensiform cartilage and umbilicus. This is joined by a third incision, which passes beneath the mamma and joins the lower extremity of the first incision. In short, a broadly oval ellipse, resembling a hen's

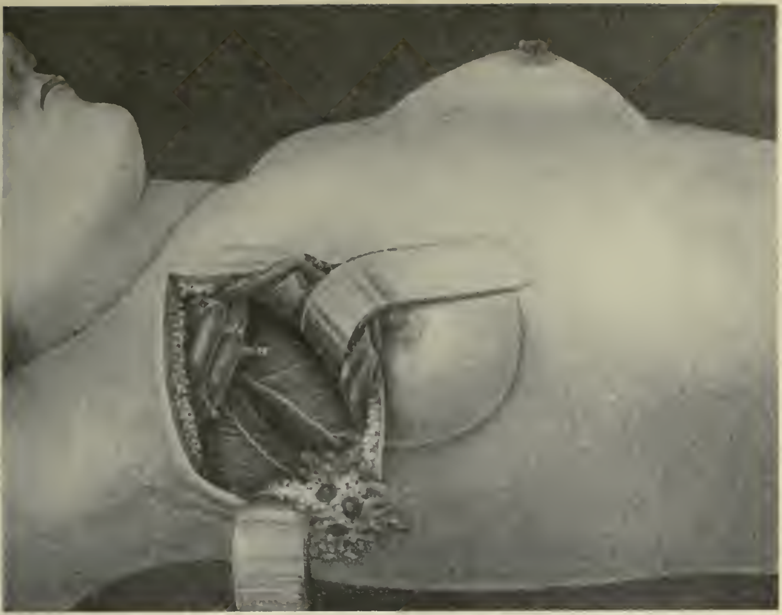


Fig. 69.—Axillary dissection completed. Fascia, fat, and enlarged glands *en masse* shown at base of axilla (Rodman, "Diseases of the Breast").

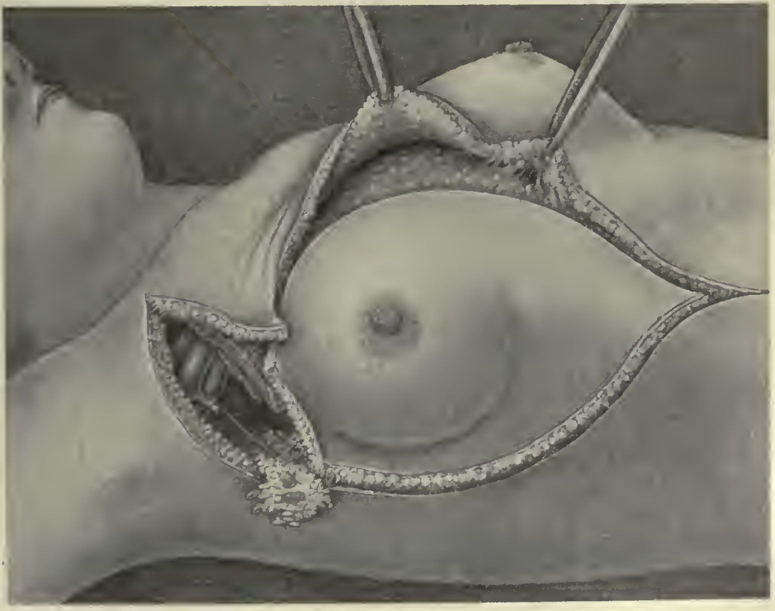


Fig. 70.—Second skin incision incircling the breast and extending well downward on the chest, to include as much as possible of the lymphatics running downward to the abdomen. Observe the extensive undermining anteriorly (Rodman, "Diseases of the Breast").

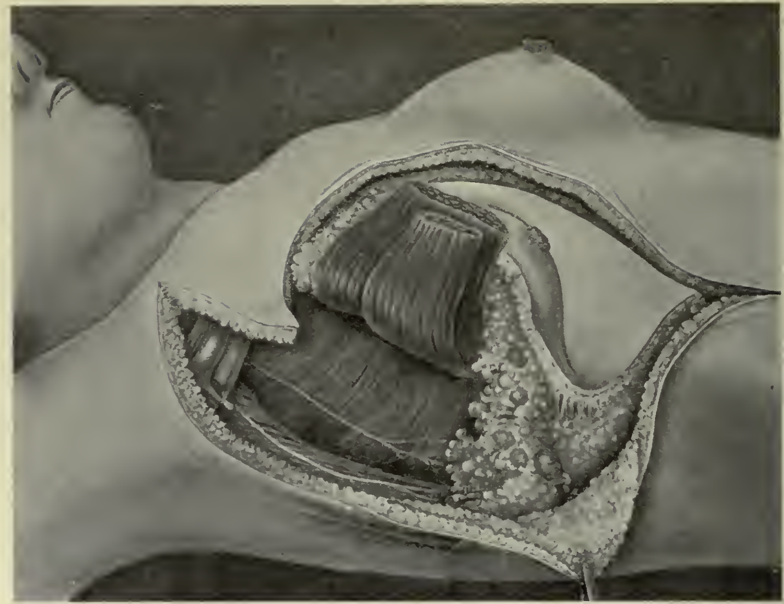


Fig. 71.—Undermining inferiorly. Muscles, breast, and axillary mass being reflected prior to removal (Rodman, "Diseases of the Breast").

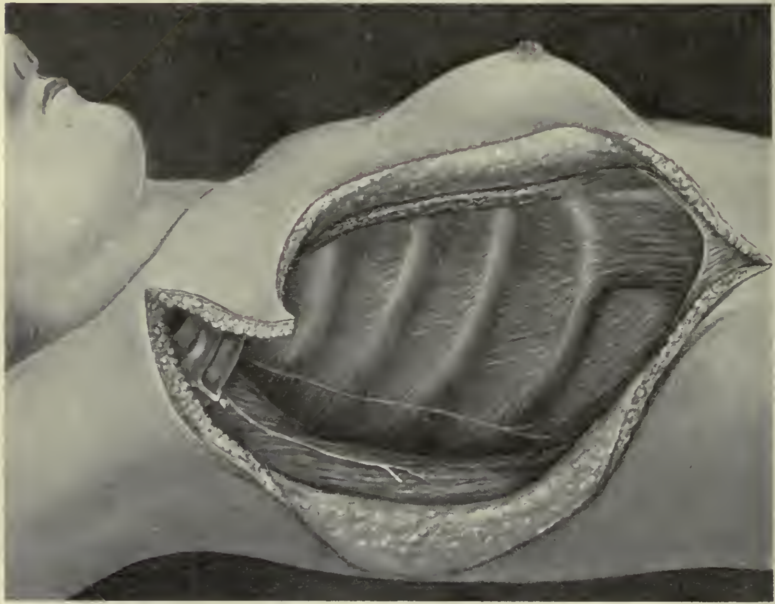


Fig. 72.—Appearance of the wound after removal of the breast muscles and axillary mass of glands and fat (Rodman, "Diseases of the Breast").

egg, is included between incisions 2 and 3. Its greatest diameter should be about six inches in a woman of average stature. We now carefully dissect the epigastric triangle, removing the sheath over both recti muscles. This is important, as Handley has demonstrated that metastases more frequently occur in the liver than elsewhere, and he believes them due to infection which travels along the deep fascial planes. Hence the reason for removing the sheaths of both recti. We have not found that it results in the slightest weakening of the abdominal wall, as hernia has never followed in any of more than 200 cases thus operated.

This done, we now remove the sheaths covering the lower portion of the serratus and upper portion of the external oblique and latissimus dorsi. It will be seen from the cuts that the knife is not carried downward, but in a slanting fashion, so as to undermine the skin in such a manner that the paramammary fat is exposed in every direction—superiorly, internally, and inferiorly, so that at least two inches of superficial fascia beyond the peripheral limits of the breast will be removed, with all contained lymph-bearing vessels. In addition to blocking the lymphatics in every direction before the breast is touched, thereby avoiding expressing cells from the tumor or its vicinity, this free removal of fat has another, and important, end, inasmuch as it facilitates and makes possible the subsequent closure of the largest wound without skin-grafting. We have not been compelled to resort to grafting in a single case since May, 1908. To say the least, it is time consuming, leaves an unsightly wound, frequently interferes with the future movements of the arm, and is undesirable if not necessary.

All lymph-bearing vessels being blocked, the breast is removed by cutting the pectoral muscles at their origin. The three perforating arteries, branches of the internal mammary, are seized at once and ligated. Also the superior thoracic, which is about the middle of the upper flap. It is surprising how little hemorrhage will occur if the operator and his assistant are both careful and alert. We have frequently operated with the loss of less than an ounce of blood, and when there has been much more hemorrhage it has been our fault. Capillary hemorrhage is perfectly controlled by the use of very hot (130° F.) water. As we no longer drain, this is a matter of importance. Suturing should not begin until hemorrhage has been stanched and all clots removed. Closure is begun at the axilla, and six interrupted sutures of silkworm-gut usually close the vertical incision. Then we begin at the abdominal end of the wound and continue until the axilla has almost been reached. At a certain point, and this is shown in the cuts, the skin from the axilla is pulled inward and so sutured as to make a triangle, thus avoiding a slight protuberance of skin which results if the suture line continues straight.

There is usually no tension at all on the suture line, as the arm can be moved freely in all directions. We make firm pressure over the axilla and chest with broad strips of adhesive plaster, so as to restrain hemorrhage, obliterate dead spaces, and insure primary union. The arm is confined for only twenty-four hours. The patient then begins to use it. Edema of the arm or impairment in its future movements gives us no concern, as they do not occur. I have deliberately resected the axillary vein eight times on account of infection and extensive axillary involvement, and in no case was there more than a slight and transient edema. In 24 cases reported to me by other surgeons the same result obtained. Edema is the result of interference with the lymphatic stream and is not due to venous obstruction. A slight and transient edema, as indicated by a puffiness over the back of the hand for a short time, is occasionally seen, and as it quickly passes away, is rather a good omen, as it shows that a complete axillary dissection has been made. I repeat and emphasize that not a single patient in more than 200 thus operated has had either permanent or disabling swelling of the arm.

Extreme shock, which we frequently encountered years ago when the vessels were not secured promptly, is unknown, for shock is practically synonymous with hemorrhage. Such patients are usually afebrile in seventy-two hours and sitting up. The sutures are removed on the tenth day, and in another day or two, nearly always, patients leave the hospital. Counting the day lost in entering the hospital and undergoing preoperative treatment, they are gone in a fortnight.

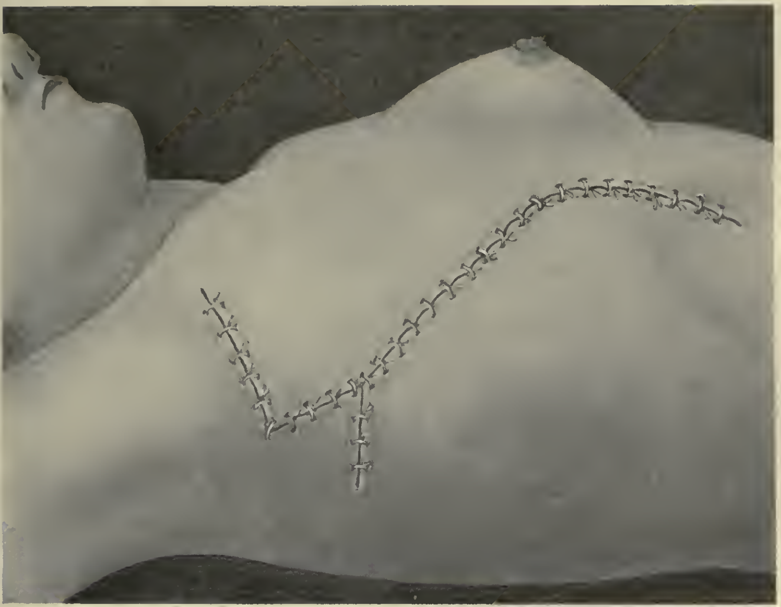


Fig. 73.—Wound being sutured. Middle third of horizontal incision may be covered, if necessary, by skin-grafting. It is rarely necessary.

I just want to say a word concerning the very suggestive statistics which Dr. Murphy has on the blackboard before us. I do not know whence they were obtained, but they do coincide accurately with my own teaching and that of Mr. Handley. Gross, and practically all the German and English teachers, taught that the lungs were more vulnerable than any other tissues in the body, so far as metastases were concerned. That was handed down and handed down until we believed it was absolutely so. Mr. Handley, in a paper read at Oxford ten years ago, where I had the pleasure of appearing jointly with him in a symposium on cancer, stated that autopsies made on a large number of patients in the Middlesex Hospital showed a majority of the metastases to be in the liver.

Dr. Murphy's table shows that, of 30 cases, the liver was involved in 13, the lung in 11, the bones in 11, and even the bone-marrow in 6. Now I insisted in the address on surgery before the Academy of Surgery in Philadelphia a year ago that I had more recurrences in the bone, particularly in the spine, than in any other part of the body. I remember to have used this expression: "To the bones belong the melancholy distinction of first place, so far as my experience goes." I do not doubt that autopsies, as generally made, will show a larger number of metastases in the liver and lungs than in the osseous system, but if we interrogated the vertebræ and long bones, made sections through them, and examined them as carefully as we do the liver and lung, I have not the slightest doubt that we would find double the number of recurrences in the bone as in either the liver or the lung.

Gentlemen, I am very much obliged to you.

Dr. Murphy: I know I am expressing the sentiment of all those present in stating that we have been entertained and greatly instructed by this very able discourse on cancer of the breast, but I regret that Dr. Rodman did not give us his opinion of ultimate results, and would ask that he kindly present his convictions on this phase of the subject.

Dr. Rodman: Just a few words as to ultimate results. They are much better now than formerly, for several reasons:

First, operations are more thoroughly done and embrace a wider zone of possibly infected tissue.

Second: Diagnoses are now made earlier and operations performed earlier, which is of much greater moment than the extent of the operation.

Third: If performed before there is involvement of the axilla, 80 per cent. and upward will be permanently cured. If there is involvement of the axillary lymphatic glands, which may occur at almost any time, but may be confidently expected within a year, the percentage of cures will not exceed 35 to 40 per cent., and not that much unless the operation is followed by a thorough course of *x*-ray treatment.

Fourth: We have, during the past five years, witnessed such excellent results, apparently, from the use of *x*-rays in advanced cases that we are now inclined to use them routinely in both average and advanced cases. In early cases their use would appear to be a work of supererogation.

Fifth: In the *most* advanced cases we prefer to treat the wound by a full dose of the rays *before it is sutured.* We have not found the time lost in so doing either harmful to the patient or to interfere in any way with primary healing of the wound, as the Roentgen laboratory is near my operating-room.

Sixth: Results in private patients are much better than in ward or dispensary patients. The former are more intelligent, have greater resistance, apply for treatment earlier, and more consistently carry out post-operative measures. Moreover, it has not been possible for me to keep track of my ward cases whose lives are a constant struggle with adversity. Of 50 consecutive private patients operated upon, 36 (72 per cent.) have passed the three-year limit, 13 are dead, and 1 has recurrence and is counted as such.

A further analysis of them shows that—

1 has passed the fifteen-year period
1 has passed the fourteen-year period
2 have passed the twelve-year period
7 have passed the ten-year period
24 have passed the five-year period
30 have passed the four-year period
36 have passed the three-year period

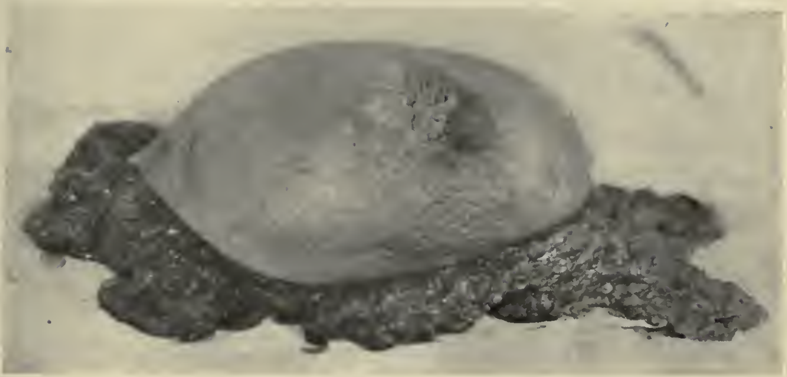


Fig. 74.—Dr. Rodman's operation for cancer of the breast. Photograph showing the gross specimen, consisting of the breast, pectoral muscles, and axillary fat, fascia, and glands removed *en masse.*

That my cases were of average severity is indicated by the fact that 2 had been pronounced inoperable by excellent surgeons. I gave them their chance. Two were ulcerating, and 6 of them required a resection of the axillary vein. Some of them were most favorable—the disease being in so early a stage that a diagnosis of cancer was impossible by clinical methods and had to be made by frozen sections. The diagnosis of cancer was confirmed by the microscope in every case, by both frozen and serial sections.

CARCINOMA OF THE COLON.—DIFFUSE MILIARY CARCINOSIS OF THE PERITONEUM. —EXPLORATORY OPERATION

HISTORY

The patient, a male aged sixty-five years, entered the hospital November 5, 1914. His family history is negative as to tuberculosis and cancer. His past personal history is negative and he denies venereal infection.

The patient's present trouble dates back to September, 1913, when he first noticed a feeling of fullness in the upper abdomen, especially marked after eating. It seemed as if he could not eat as much as formerly—as if his appetite was too easily satisfied. This condition continued, and, in addition, he became severely constipated, often going from two to five days without a bowel movement. There has been no blood, slime, or mucus noted in the stools. Gradually some tenderness developed across the upper abdomen, especially in the left hypochondrium, together with a feeling of fullness there, which seemed to have a burning, gnawing sensation connected with it. He never vomited in those days. The pain was at its worst usually about one-half hour after eating; from then on it would diminish to the original mere feeling of fullness.

Six months ago his condition became worse and he often had a feeling of nausea, which came on from three to four hours after eating. The burning sensation in the left hypochondrium gradually became more marked, and the patient became very constipated and suffered much pain in the lower abdomen. He never vomited until two or three weeks ago. The occasion of his first vomiting was when his stomach was washed out. After the first performance of this procedure he immediately vomited. No blood appeared in the vomitus. The patient has lost 30 pounds in weight in the past twelve months. He thinks that he becomes nauseated because his saliva shows a tendency to accumulate. Probably this is merely the usual ptyalism accompanying and preceding nausea.

COMMENTS AND OPERATION

Dr. Murphy (November 7, 1914): What does the blood count show?

Intern: I have no record.

Dr. Murphy: What about the feces?

Intern: Blood is present, and much mucus.

Dr. Murphy: What is the matter with him?

Intern: He has a feeling of fullness coming on after eating.

Dr. Murphy: How soon after?

Intern: Immediately after eating. That was his complaint at first. Six months ago he became worse and developed a feeling of nausea, which came on rather constantly about three hours after eating. He did not vomit, but was very constipated.

Dr. Murphy: Yes. What is the matter with him?

Intern: The pain seems to be most marked in the left hypochondrium. I do not know what is the matter with him unless it is some involvement of the colon in the neighborhood of the splenic flexure.

Dr. Murphy: What makes you think so?

Intern: I should judge that the stomach is not involved because the history is not typical for a gastric lesion.

Dr. Murphy: And yet he says that the pain in the upper abdomen and the gas formation used to come on half an hour after meals and sometimes nausea and vomiting occurred at about that same time? The recent manifestations, consisting of nausea and salivation, now come on when?

Intern: Three, four, or five hours after eating.

Dr. Murphy: What does the patient vomit now?

Intern: He has vomited only twice recently—once after his stomach was washed out at home and again after he was in the hospital.

Dr. Murphy: What has been his greatest complaint?

INTERN: Constipation.

DR. MURPHY: Inability to get his bowels to move with what? What do you mean by constipation?

INTERN: Inability to get the bowels to move without the use of enemas.

DR. MURPHY: This patient could not get his bowels to move even *with* the use of enemas. Constipation is inability to get the bowels to move without the aid of cathartics. This condition is more. This patient is unable to get his bowels to move with the aid of either cathartics or enemas, which means something more than constipation—something entirely different, in fact. What else suggestive do you find in the history? What did he say about the rumbling in his bowels?

INTERN: Nothing to me.

DR. MURPHY: One of the first questions to ask such a patient is, "Have you much rumbling in the bowels?" This patient volunteered that information to me. He complained of the "enormous noise being made in the stomach by the gas." Why? Because where there exists a gradually increasing obstruction of the bowel, intestinal peristalsis keeps increasing and increasing in intensity as the hypertrophy of the muscular layer of the bowel increases in trying to force the intestinal contents past the obstruction. From two and one-half to four hours after meals this patient starts to have increased peristalsis. The higher in the bowel the obstruction lies, the sooner after a meal does a patient develop this manifestation. In examining a case suspicious of intestinal obstruction the surgeon should always use his stethoscope. He may thus be materially aided in diagnosing not only the existence of an intestinal obstruction, but also its location, for the borborygmi will usually be most intense in the immediate vicinity of the obstruction. In using the stethoscope for this purpose one must always bear in mind that the previous administration of morphin or opium practically nullifies the value of the examination, because these drugs stop the peristalsis. Of course, when the peristalsis has subsided for some reason or other in an acute or chronic obstruction the stethoscope does not have much diagnostic value. Its use then is deceptive, rather than instructive. It is the positive findings of the stethoscope, not the negative findings, which are of value. What one hears typically in an obstruction is a rumbling noise, which gradually increases in intensity as the peristaltic wave approaches the seat of the obstruction, and ends with a sudden roar as the obstruction is reached. Then there is silence for a short time, and the occurrence is repeated. I recall a recent case of obstruction with very marked borborygmi. She came for operation some five or six days after the first active manifestations of the obstruction developed. We kept her here under observation for two weeks before we operated. She was not in any way an urgent case. We gave her a meal of meat and then brought her into clinic four hours later. The borborygmi were so marked that one could turn his back to the patient and yet hear her peristalsis anywhere in the clinic room. When we operated we found a constriction of the annular type in the lower portion of the ileum.

Our patient of to-day apparently has two separate pathologic conditions, one of which is probably the sequel of the other—a gastric lesion, either functional or organic, and a mass in the left hypochondrium, producing a chronic obstruction of the large bowel near the splenic flexure. The question to be decided is whether we have to deal with a gastric ulcer or cancer which has perforated into the intestine and produced more or less perigastritis and pericolitis, or whether it is a carcinoma of the sigmoid flexure which has become adherent to the stomach, so that a full meal produces pain by pulling on the adhesions?

Judging from the patient's history and symptoms, the condition is much more likely a carcinoma of the splenic flexure now involving the stomach than ulcer or cancer primary in the stomach. In the first place, the patient's earliest and most prominent complaint has been his intractable constipation. Then he developed the feeling of fullness in the upper abdomen after meals. The higher up a chronic obstruction lies in the bowel, the earlier will it give rise to nausea and vomiting; and the lower down it lies, the longer will a sensation of fullness in the upper abdomen be the only gastric symptom, and the longer will the appearance of vomiting be delayed. With rectal cancer, nausea and vomiting are very late symptoms, and, therefore, have relatively little diagnostic value.

Secondly, if this is primarily a gastric condition, the lesion is certainly not located at the pylorus, because there have been no symptoms of pyloric obstruction—dilatation of the stomach, visible peristalsis from left to right, vomiting of large quantities of gastric contents, and pain promptly relieved by vomiting; and the pyloric region is the most frequent site of gastric ulcer and cancer.

Thirdly, if this lesion were an ulcer or cancer of the fundus or greater curvature of the stomach perforating into the splenic flexure, and of sufficiently long standing to produce the large tumor mass now present, one would expect the patient to show a high grade of cachexia as the result of the short-circuiting of a considerable portion of the ingested food from the stomach to the colon, thus escaping the digestive and absorptive action of the small bowel. But the patient still shows an excellent degree of nutrition, although he has lost about 30 pounds in the last year.

Fluoroscopy after a bismuth or barium meal might clear up the diagnosis, and then again it might not, and might only add to the obstruction in the bowel. An exploratory operation will permit us to make the diagnosis without further ado, and at the same time will permit us to carry out such remedial measures as may still be possible—a gastrojejunostomy, if the condition is an ulcer of the stomach, or a short-circuiting operation, transverse colon to sigmoid, if we find an inoperable cancer of the splenic flexure.

[Dr. Murphy opened the abdomen through a left paramedian incision in the epigastrium, and then explored the peritoneal cavity.]

Here is a hard, woody mass in the splenic region, probably carcinomatous. We will make another large incision at right angles to the first incision, across the rectus muscle and following the line of the transverse colon. These incisions will give us ample room to do anything we desire, and will allow us, if necessary, to make a colostomy here in the epigastrium—a very favorable place for it. If conditions are not thoroughly favorable for a short-circuiting operation, we shall perform a colostomy of the transverse colon instead. We shall not make it, however, until we determine definitely whether the carcinoma can be taken out or not. We always fasten towels on the wound so they overlap the edges of the skin. Thus there can be no hand-contacting with the skin after the second incision has been completed. Now that we have reached the peritoneum, we will clean up again and then are ready to go into the abdomen.

[On opening the peritoneal cavity a moderate amount of serofibrinous fluid escaped through the wound.]

Let the record show that the fluid is not bloody. Hemorrhagic ascites is the type which is most characteristically associated with a malignant neoplasm.

The first feature that attracts one's attention in this field is the appearance of the small intestine. It does not come up into the wound because of multiple small carcinomata all over its surface—these little white areas which have formed adhesions with the posterior parietes and hold the bowel bound down. The entire splenic flexure of the colon is bound down into a hard mass to which a loop of small intestine is also adherent. The stomach is only superficially involved in the mass. Therefore we can now decide with ease that the primary growth was not in the stomach, but in the large bowel.

In view of the presence of this extensive carcinomatous peritonitis (see the accompanying Fig. 75) I shall try to do neither an excision nor a colostomy. With this general peritoneal carcinosis involving every portion of the peritoneum, and feeling like masses of small shot all over the intestine, as the examining finger touches them, it is no use to bother the patient with more surgery. We would not prolong his stay in this world or his comfort while here by making a more extensive wound for cancer metastases to develop in.

We will close this abdomen with a single row of figure-of-8 silkworm-gut sutures. The lower loop of this 8 includes the peritoneum. We are using through-and-through stitches in place of a layer closure with catgut, so that we can let this man

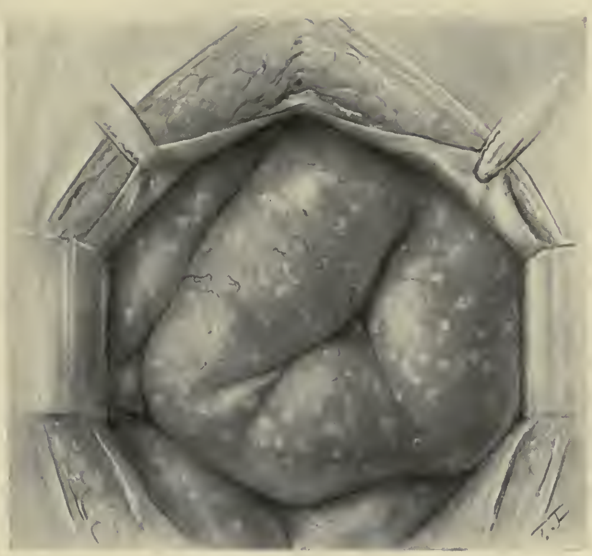


Fig. 75.—Diffuse miliary carcinosis of the peritoneum going out from a carcinoma of the colon near the splenic flexure.

up out of bed right away and diminish his liability to post-operative pneumonia and thrombosis.

The lesson that we should learn from the case is this: The man has been complaining of his abdominal trouble about one and one-half or two years. He has had this neoplasm growing in his colon all that time; and, finally, when he reaches this superlatively hopeless condition, where nothing can be done to save his life, he is brought into the hospital for treatment. His doctor very frankly stated that he believed nothing could be done for the patient. This patient is the victim of procrastination. He himself is principally to blame, for he declined exploratory operation at first. But his doctor is not altogether guiltless, for he recognized this as a serious trouble a long time ago, and had he *insisted* and *insisted* and *insisted* on an operation, and had he then refused to take care of the case further unless an operation was performed, the patient would have consented to this step at an earlier date, and so perhaps have had a chance for his life.

I want to call your attention particularly to this analysis of cancer statistics which I have had placed on the blackboard. Of 56 cases of carcinoma of the intestine that went to autopsy in the Virchow Pathologic Institute in Berlin, practically one-third had no demonstrable metastases. If one-third of them had no metastases anywhere in the body even at the time of death, how many would have had no metastases a year, a year and one-half, or two years before death? Certainly, more than one-third. That means what? That more than one out of every three of these patients died unnecessarily. Will any one here deny that had these patients been promptly and radically operated as soon as it was possible to make the diagnosis of the trouble one-half of them would have been saved? That fact demonstrates the heavy obligation which rests upon our profession, and which our profession is not meeting adequately at present. *We must make early diagnosis of cancer. We must insist on early operation*—not merely recommend operation, but insist on it and compel it.

Carcinoma occurs much more frequently in the large intestine than in the small gut. I do not know just what the percentage is, but I should estimate that it occurs about 30 times in the large intestine, if not more, judging by my own experience, to the once in the small intestine. Why do these intestinal cancers have so few metastases? Because the large intestine, with the exception of a few portions, such as the splenic flexure, the hepatic flexure, and the junction of the sigmoid with the rectum, has a sparse lymphatic supply. These three portions which I just mentioned have a richer lymphatic supply, and therefore cancer at these sites gives a worse prognosis than elsewhere in the bowel.

A somewhat similar rule holds for carcinoma of the stomach. Carcinoma of the fundus of the stomach has but a very small percentage of metastases. Why? Because of the condition of the lymphatic supply in that area. Because the sparsity of the lymphatics in the fundus of the stomach, as in the fundus of almost any hollow viscus, makes it a relatively safe position in which to have a carcinoma develop. That is also true of the fundus of the uterus. For example, I operated on a patient fourteen years ago for a carcinoma of the fundus of the uterus. It was the last day that I was on service at the College of Physicians and Surgeons in Chicago. So rotten was the fundus of that uterus that when I put on the volsellum to lift up the organ the fundus pulled completely off. Then I had to go in and fairly grub out the rest. But that woman is living yet.

On the other hand, I have at present not a single referred case of carcinoma of the cervix of the uterus operated by any method who is living two years since operation without evidence of return. That statement illustrates the difference in prognosis between a fundus carcinoma and a cervix carcinoma. The same rule holds good for cancer in the fundus of the stomach, the fundus of the urinary bladder, and the fundus of the gall-bladder. It is true only because the fundus of all organs possesses a sparse lymphatic supply. The large intestine falls in just that same class. But this patient to-day has not only a large local growth, but also a rupture and exfoliation of the cancerous material into the free peritoneal cavity, with miliary metastases all over the peritoneal surface. In about 3 per cent. of cancers of the stomach in women one finds, on examining Douglas' pouch, that it contains secondary carcinomatous deposits, due to cancer-cells that have escaped from the lymphatics and dropped down through the peritoneal cavity into Douglas' pouch. W. J. Mayo was the first American surgeon to call attention to this particular pathologic condition in a forceful way.

[Note.—The abdominal wound healed by complete primary union. The patient went home after a two weeks' stay in the hospital and has not reported to us since then.—Ed.]

EPITHELIOMA OF THE UPPER LIP STARTING IN AN OLD LUPUS SCAR.—EXCISION, PLASTIC CLOSURE

HISTORY

THE patient is a married man aged forty years. His family history is negative. He had the usual diseases of childhood. At the age of seven he had an attack of typhoid fever. He had tonsillitis at the age of twenty-five, and at this time his nose and throat were cauterized. He had "nervous prostration" six years ago, and did not work for four months.

Two years ago, in 1912, the patient noticed on his upper lip, at the left side, a spot, ⅓ inch in diameter, which resembled an area that had been burned. This spot formed a sore, which looked for a while like a fever-blister, but finally began destroying the skin. He paid little attention to it until about four months later, when he noticed that the sore had taken on a tendency to destroy more tissue in the depths of the ulcer, although it was not enlarging much laterally. He then took *x*-ray treatments for six months, and at the end of this time the ulcer apparently healed. He never had had any pain from it.

Two weeks later, on scratching the spot, he noticed a dry scaling over it. He took *x*-ray treatments again for six weeks, from October to December, but without improvement this time. It was no better by February, 1913, and so, on February 12, 1914, he had a Wassermann test made, which was reported positive. He then took specific treatment for six months, consisting of salvarsan and mercury injections and the iodids by mouth. Another Wassermann reaction four months later was negative. Since then the ulcer has grown a little deeper, and is now ⅛ inch in depth and ½ inch in diameter. It is much indurated around the edges, and the lip is puckered up near the left labial angle by the contraction of the cicatricial tissue in the base of the ulcer and around it. There is never any pain in the sore, but there is a sharp pricking sensation present in the skin for about ½ inch outside the circumference of the ulcer. There are no enlarged glands palpable in the neck.

COMMENTS AND OPERATION

Dr. Murphy (September 12, 1914): This is a very interesting case—interesting and instructive, because this small sore has a complicated story, which is not properly brought out in the intern's history, and which made the diagnosis not altogether easy, just when an early diagnosis was extremely important in order to keep the patient out of grave danger—the danger of losing his life by the spread of this cancer. In the first place, the point of origin of this lesion is not correctly stated. It began at the left nasolabial angle, not on the lip. What lesion did the patient have at this spot, doctor, before the present "sore" began?

Intern: The history does not state.

Dr. Murphy: But it *should* state. When did the present sore begin?

Intern: Two years ago.

Dr. Murphy: What were the previous illnesses?

Intern: A cauterization of the nose during an attack of tonsillitis; typhoid at seven years of age, and the diseases of childhood.

Dr. Murphy: That history is worthless, so far as it throws any light on the genesis of his present trouble. It is 20 per cent. worse than nothing, for it leaves us in worse darkness than when we started, because of a false statement in it. It leads us to believe there is no clue, although the clue is obvious and easy to follow.

When the patient was twelve years of age he had a lesion at this very point which was many months in healing. Finally it healed, with an elevated hypertrophic scar, that looked like the scar of a burn. This small keloid was irritated and frequently cut in later years with his razor in shaving. Twenty-five years passed, and then a slight, superficial erosion began to appear at the center of this scar. The erosion grew slowly deeper and deeper. Finally the patient was put under *x*-ray treatment. The ulcer then healed over, but in a short time broke down again. *X*-ray treatments were again used, but this time without avail, which is the rule in all courses of *x*-ray and radium treatment after the first. A Wassermann test was made with the thought that the diagnosis of epithelioma might be an error. It was positive. The patient, accordingly, was put under antiluetic treatment. The ulcer showed no more tendency to heal under this specific therapy than it did under the second application of the *x*-ray.

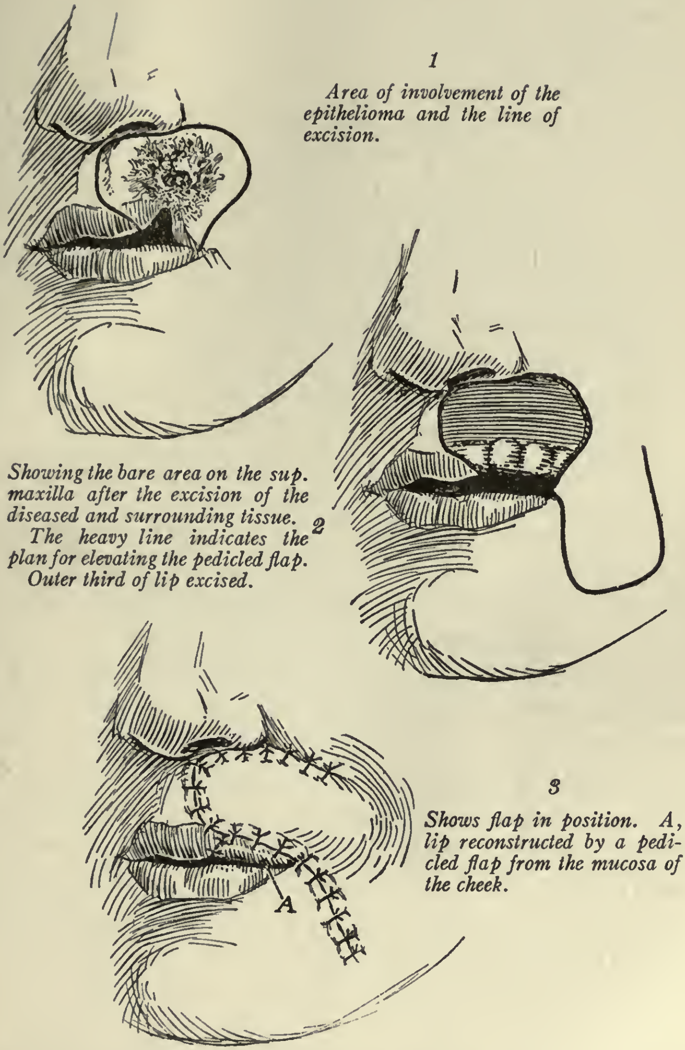
Is not that an entirely different story from the one the doctor read us? The original lesion occurring at the age of twelve was undoubtedly a small lupus vulgaris focus, from the patient's description of it. It healed spontaneously in the course of a few months, as small lupus lesions more often do in our country than abroad, and the patient remained perfectly well except for the hypertrophic scar—a small post-lupoidal keloid. The patient denies knowledge of any syphilitic infection, his skin and mucous membranes show no trace of syphilitic residua, and the examination of his wife and children is likewise negative. Therefore the diagnosis of syphilis should still be considered questionable, notwithstanding the single positive Wassermann. The patient was given three salvarsan injections besides mercury rubs, but they seemed to have no influence on the course of the lesion. If this lesion is a recurrence of his lupus, eroded and infected by repeated shaving,—and lupus does not ulcerate easily,—it should have been healed by the *x*-ray, as so many of them are in the hands of capable dermatologists. If it was a gummatous ulcer, it should have healed under the combined salvarsan and mercury treatment. Since we can, therefore, rule out tuberculosis and gumma with a very considerable degree of certainty, we are driven to the inevitable conclusion that this lesion is just what it now and previously has always appeared to be—an epitheliomatous ulcer developing in an old lupus scar.

Is it rare to have an epithelioma start in an old lupus scar? No, not at all. We see it in this country not infrequently, and still oftener abroad, where, especially in the large cities, lupus appears to be of more frequent occurrence than here. In the foreign dermatologic museums one sees many beautiful reproductions of this type of growth. The Baratta collection of dermatologic *moulages* at the Hôpital Saint Louis in Paris; the collections made by Henning for the Riehl and Finger clinics in Vienna; and the Neisser collection at Breslau—all have many examples of "*epithelioma in lupo*," as they usually label it.

Is epitheliomatous degeneration of lupus scars becoming more common? I think so, and most dermatologists with whom I have talked on the subject agree with this view. Why is it becoming more frequent? I believe it is due to the increasing frequency of the use of the *x*-ray in lupus treatment. The cases treated with the Finsen light or by curetment, followed by pyrogallic acid cauterization, do not show nearly the same inclination to epitheliomatous degeneration that the roentgenotherapy cases do.

Is this peculiar susceptibility of lupus scars to the deleterious effects of the *x*-ray the reason that prolonged *x*-ray treatments failed to cure this cancer, whereas they are usually very successful in the treatment of basal-cell epitheliomata in this location? I cannot answer this question with complete assurance, but this explanation seems to me plausible and probable.

In the two years that this ulcer has existed it has already eaten down through the patient's lip to the periosteum of his superior maxilla; and the contraction of the stroma of the tumor has drawn up the angle of his lip (as shown in the accompanying illustrations). He has no evidences of regional or general metastases, so far as we have been able to determine. Has the *x*-ray treatment done the patient any harm? No, I think not. Rather, it has been of considerable value to him, since it has produced a marked scar-tissue reaction in and around the tumor, and has delayed and circumscribed its extension by scar-tissue blocking of the natural paths of cancer progress, the lymph-channels, and the tissue spaces. Because of this circumscription of the growth we shall not have to excise so wide an area of tissue around the growth as we otherwise should have had to. This is



1

Area of involvement of the epithelioma and the line of excision.

2

Showing the bare area on the sup. maxilla after the excision of the diseased and surrounding tissue.

The heavy line indicates the plan for elevating the pedicled flap.

Outer third of lip excised.

3

Shows flap in position. A, lip reconstructed by a pedicled flap from the mucosa of the cheek.

Fig. 76.—Epithelioma of the upper lip starting in an old lupus scar. Excision. Plastic closure of the defect with formation of a new vermilion border to the lip by bringing down a small flap of buccal mucosa.

Handley's interpretation of the action of the *x*-ray on these growths, and is, I believe, the correct one.

[Dr. Murphy then excised the tumor down to and including the periosteum and mucosa, cutting through normal skin on all sides of the tumor, from ¼ to ½ inch from the tumor margins, as shown in the accompanying illustrations. The angle of the lip was preserved, so as to get a good vermilion border for the new lip. This was the only part of the plastic flap used about which there could be any question as to viability. The mucous membrane of the cheek was swung forward as a pedicled flap to fill up the gap in the mucosa. The pedicled skin-flap used for the plastic to fill the operative defect fitted perfectly into place, and ought to survive *in toto*.—Ed.]

Remember that in operating in an area so vascular as the lip one can have hand-contacting and almost foot-contacting with the wound and yet not infect it. It is an entirely different proposition from surgical work in a joint.

We are placing no dressing at all on this wound. We are leaving it perfectly dry, with only the red bismuth subiodid powder on it. So long as we keep the wound dry and covered with this desiccating powder the wound will remain as clean and secure as under a heavy, bunglesome dressing; more so, in fact, since it is hard to keep the latter free from vomitus, saliva, and food in this location; whereas without the dressing the soiling of the wound, if it occurs, is at once recognized, the lip washed off with alcohol, and redusted with the bismuth powder.

[Note.—The cosmetic result is excellent and the mobility of the lip is good. The hair-growth on the transplant is sufficiently thick to allow the patient to grow a mustache and thus cover over the linear scar on the lip.—Ed.]

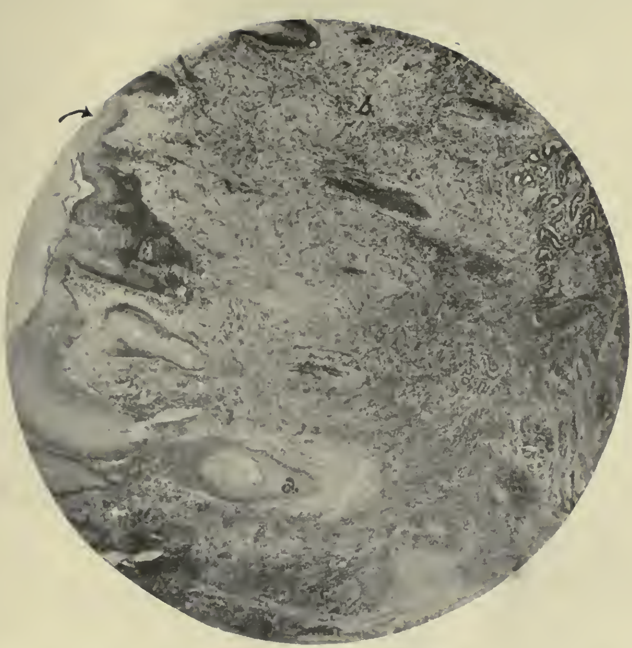


Fig. 77.—Basal-cell epithelioma of the upper lip beginning in a lupus scar. The arrow indicates the point of transition from the thin epithelium of the scar to the greatly lengthened epithelial column (*a*) at the border of the epithelioma. Note the subepithelial connective tissue of the lupus scar (*b*) and the compressed atrophic remnants of the cutaneous glands in it lying to the right of the field (preparation made by Dr. W. O. Sweek).

INTRAMURAL FIBROID OF THE UTERUS.—DIAGNOSIS.—HYSTERECTOMY

HISTORY

The patient, an unmarried woman thirty-two years old, entered the hospital October 1, 1914. She has had the usual diseases of childhood. Her family history is negative as to tuberculosis and carcinoma.

She began to menstruate at the age of fourteen years, regularly every twenty-eight days, five days in duration, using from eight to ten napkins a day up to 1910, when her periods began to occur more frequently, coming on every two or three weeks, with an increase in the amount, but not in the duration, of the flow, which remained of the five-day type. She has had no pain worth mentioning with menstruation at any time, and no continuous flow, and yet she has become very weak and anemic, apparently solely because of the increased amount and frequency of the flow. She sometimes has a two weeks' interval and sometimes a three weeks' interval between periods, never going the full four weeks since 1910. She has had no intermenstrual flow, and no gush of blood at any time. She often passes clots. Her last period was October 2, 1914. In April, 1914, she noticed for the first time a tumor mass in the midline of the lower abdomen. It felt very hard and has gradually increased in size.

COMMENTS AND OPERATION

Dr. Murphy (October 19, 1914): Now let us see exactly what you mean in regard to the time intervals between the patient's periods. Did you measure the two or three weeks' interval from the beginning of one period to the beginning of the next, or from the end of one period to the beginning of the next?

Intern: She would have a period and two weeks after she would have another period.

Dr. Murphy: Two weeks after what?

Intern: Two periods a month, doctor.

Dr. Murphy: Why do you not make it clear? These fibroid patients usually have prolonged periods. You must state definitely that the interval is two weeks from the beginning of one flow to the beginning of the next, if that is what you mean. Your history will not aid in making an accurate diagnosis unless all its details are accurate, particularly the details of so important a matter as the duration and character of menstruation. Patients with uterine fibroids may be menstruating every two weeks, and yet have only two or three days' interval between the end of one flow and the beginning of the next; or the flow may be practically continuous, one period merging into the next. You have also failed to state how many napkins the patient is using daily at present. I remember from the history which she gave us in the office that the duration of menstruation, five days, has not changed since 1910, but that her periods have become more frequent and the amount of her flow has increased. I noted this fact because it constitutes an exception to the general rule that with a myofibroma not only is the quantity of flow increased, but also its duration.

We are trying to check up all our uterine fibroid cases with the Abderhalden test, in order to get at its practical value in these cases. The average obstetrician cannot check up the practical value of the Abderhalden test in such work, because although he has sufficient obstetric material at his command, he lacks the surgical material, the tumors of the pelvis and lower abdomen in which the Abderhalden test has its greatest differential value. Therefore, I believe, it is the surgeon who will ultimately decide the value of the test, and from that point of view I am having the Abderhalden test made in every case of uterine fibroid which comes to me.

Let us analyze the history of this patient. She is only thirty-two years old. That is an early age for a fibroid to produce such disturbances of menstruation, and twenty-eight years, her age in 1910, four years ago, when her menstrual irregularity began, is a still more unusual age for a fibroid to produce symptoms.

The rule is that fibroids do not commence to manifest themselves by menorrhagia until the patients are at least thirty-five years old; and from that age up to fifty years fibroids make their presence manifest with increasing frequency.

How does a fibroid disturb menstruation? It does it in a regular and systematic way. First comes the increase in the quantity of the flow; second, the increase in the duration; third, the patients practically never have an intermenstrual flow; fourth, the menopause is postponed by the tumor; the fibroid patient at forty-eight, forty-nine, and fifty years of age frequently is still menstruating; fifth, patients with a fibroid practically never have the flow subside for one, two, or three months, and then commence again. When such patients come to their late climacteric, the flow *suddenly* stops. With the normal menopause which comes at forty-five to forty-six years, as a rule, the flow gradually diminishes in amount and duration and finally subsides; the woman then may skip a month or two, but flow slightly again before she is through for good and all. When a fibroid patient, however, reaches her delayed climacteric, although she may have been flowing copiously and regularly up to that time, once she skips a month she does not flow again, as a rule. She is permanently through.

This sudden cessation of menstruation is a guide to the diagnosis of the tumor, so that when one hears from a patient that she has been menstruating up to fifty-four, fifty-five, or fifty-six years of age, regularly and uniformly, and then stops suddenly, one suspects at once that she has either an intra-uterine polyp or a fibroid. So common is the postponement of the menopause with a fibroid that we usually state that a patient who is menstruating at the age of fifty in the regular, uniform way has 65 per cent. of probability that she has a fibroid. This probability increases about 5 per cent. for each year she is over fifty, so that a patient who is menstruating at fifty-five regularly has a 90 per cent. probability of possessing a fibroid or a uterine polyp. Beyond fifty-five years the annual percentage increase is not so large. Thus you see the menorrhagia of uterine fibroids is a regular flow, not an intermittent one.

The maintenance of a regular flow speaks strongly in favor of the diagnosis of fibroid and against carcinoma. A patient who has her menstruation cease at forty-five years and subsequently develops a flow at the age of fifty-five, equal to or even greater in amount than her normal menstruation was, has 95 per cent. of certainty that she possesses a uterine carcinoma, regardless of what the result of the doctor's bimanual examination may be. That difference between the clinical histories in these two classes of cases is of great diagnostic value.

When was this woman's last menstruation?

INTERN: September 2d.

DR. MURPHY: That statement is not correct.

INTERN: I beg your pardon; it was October 2d.

DR. MURPHY: That is correct. I should not open her abdomen if, with a fibroid, she had nevertheless missed a menstruation, because in that event she might be pregnant. That is the last question to ask in one's history-taking, the date of the last menstruation, for one asks it as a check on the rest of the history.

We have not made the Abderhalden test here. It was ordered and the order was overlooked. If the intern's first statement as to the date of the patient's last menstruation were correct, September 2d, we should have insisted that the Abderhalden test be made, and even had it turned out negative, we should not have operated. The clinical fact of a missed period would have outweighed this laboratory test in diagnostic importance.

When I made the bimanual examination on this patient I found it difficult to reach the cervix, even with my long fingers. It has passed up under the symphysis. The uterus was enlarged to about the size of a two or three months' pregnancy; but it was hard in consistence, and the vaginal portion was also quite firm. I get the history of these patients and examine them all myself in the office, and by so doing avoid many mistakes and oversights. It is my system. No matter how careful one may be in an individual case, one must have a system which cares equally well for all cases. It is surprising how often a good system, carefully adhered to, keeps one out of trouble. One is

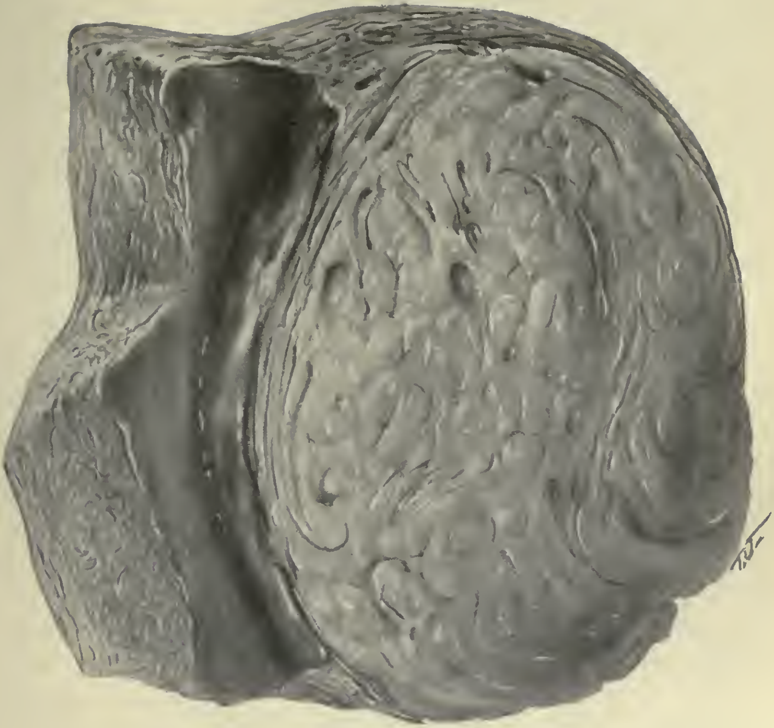


Fig. 78.—Intramural fibroid of the uterus in a woman of thirty-two years, causing profuse menorrhagia, which nearly exsanguinated the patient. The specimen, removed by amputation of the uterus at the cervicocorporal junction, has been split open to demonstrate the intimate relation of the tumor to the uterine mucosa.

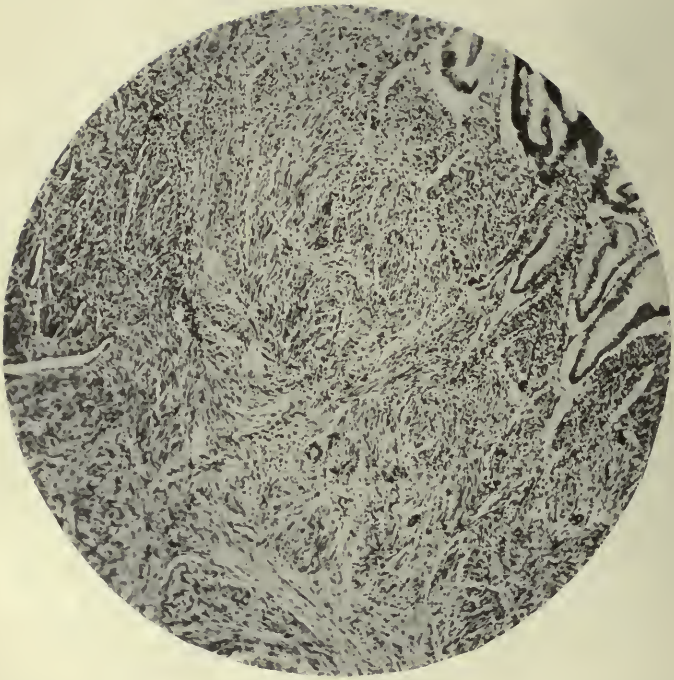


Fig. 79.—Intramural fibroid of the uterus. The photomicrograph shows the myofibroma lying directly beneath the uterine glands. The submucous plexus of vessels does not appear greatly dilated in this specimen (prepared by Dr. W. O. Sweek. Magnification, 60 diameters).

certain to make mistakes sometimes; but careful, persistent, routine work keeps them down to a minimum.

[Dr. Murphy made a right paramedian incision, going through the right rectus muscle about one inch from the midline. Since the tumor was an intramural fibroid and very large, Dr. Murphy did not feel justified in making an attempt at enucleation, so he amputated the uterus at the cervicocorporeal junction from behind forward in the usual way, which has been previously described in the CLINICS. Both ovaries and tubes were left *in situ*.]

Let the record show that the fibroid involved almost the whole of the fundus and elevated the fundus in the intertubal line; that the round ligament seemed to come over with the tubes, just about the middle of the tumor.

Let us examine the specimen. It is a large, typical, intramural fibroid. One now can see why this woman had such a profuse flow that she was incapacitated for a useful life, and why her skin is of such a peculiar yellow color, something like the yellow which one sees in a case of pernicious anemia—lemon-yellow, we usually call it. This color is not infrequently seen in the severer grades of secondary anemia, as well as in the so-called primary anemias. She has flowed so much because the tumor lay just beneath the uterine mucosa, where its interference with the blood-supply of the mucosa is at its maximum. This gross specimen is very interesting when considered in connection with the clinical history, because it explains the patient's symptoms so completely.

[NOTE.—The wound healed by complete primary union. The patient ran an afebrile course to complete recovery. She was allowed to get up on the sixteenth day after the operation.—ED.]

HYPERTROPHY OF THE PROSTATE.—URINARY RETENTION AND SELF-CATHETERIZATION.—CYSTITIS, PERIPROSTATITIS, WITH MULTIPLE ABSCESS AND FISTULA FORMATION.—PERINEAL PROSTATECTOMY

HISTORY

The patient, a married man aged sixty-six years, entered the hospital December 4, 1914. His past and family histories are negative. He denies venereal infection.

His present trouble began in 1910. The patient first noticed a slight burning sensation at the beginning of urination. Three months later he noticed that, in addition to this burning sensation, it was difficult to start the stream. He had no dribbling then. During 1911, when the patient was exposed to cold or wet his feet, he had to go to a doctor to be catheterized, because at such times voluntary urination became altogether impossible. This happened, however, only two or three times during the year 1911. As a rule, he urinated frequently, but only a small amount at a time. In 1912 he found it necessary to be catheterized six times during the year. In 1913 his condition grew still worse, but still it was only during the winter months that catheterization was necessary.

On December 24, 1913, he was taken with a dull pain and a feeling of fulness, more around than in the rectum. He suffered intense pain on sitting down. He also had a slight degree of fever, he thinks, but he does not know how much. On December 25th he felt somewhat worse, but was not confined to bed. On December 27th he still had the same dull pain, and, in addition, a high fever. A doctor was called, who ordered the patient put to bed. The perineal region and the left side of the scrotum were much swollen at this time. One week later pus began to discharge from the rectum, and the pain was relieved. Pus continued to discharge until February 1, 1914, and then the sinus healed.

In March, 1914, he was taken to a hospital for further treatment of his bladder trouble. He stayed there one week. After going home he was up and about again, and gained both in weight and in strength. In March, 1914, an abscess formed in the right groin. Although it was often poulticed and kept enlarging, it was not opened until August 4, 1914. It has been discharging ever since, but the discharge seems now to be decreasing in amount. There is still some soreness present around the sinus. Urine has leaked out through this second abscess cavity ever since the abscess was opened. The patient states that it is this urinary leakage which necessitates catheterization at the present time because he now passes his urine without pain or burning. There is also a perineal fistula.

Examination, December 8, 1914: There is a continuation of the cicatricial tissue surrounding the prostate, from the prostate over to both sides of the pelvis. The prostate itself is woody hard. The phenolsulphonephthalein test shows a total excretion of only 35 per cent. in the first two hours, instead of the normal 55 per cent. or 60 per cent.

COMMENTS AND OPERATION

DR. MURPHY (December 9, 1914): The difficulties with which I have to contend here are not inconsiderable. They make this a very unlovely and undesirable case. First, the cellular tissue on both sides of the pelvis is densely infiltrated, and the infiltration further involves the deep structures of the perineum. Second, he has had a fistula due to the opening of a periprostatic abscess into the rectum. This fistula is now closed, but, as its sequence, the capsule of the prostate is organically united to the rectum. The presence of this firm organic adhesion is a considerable danger to the patient, since, because of it, in grubbing out the prostate, we may tear into the rectum. Third, it is going to be difficult to recognize the outer margin of the prostate when we reach it, because this dense infiltration is present on both sides of the gland, and merges with it. All this disagreeable mess which makes our operation so much more difficult and dangerous than usual is the more regrettable because so easily avoidable. It is all chargeable to the patient this time, and not at all to the doctor. The doctor who had charge of the patient when he was first catheterized told him that he should have an immediate operation,—a prostatectomy,—for the removal of the urinary obstruction. That was correct. The day a man with an enlarged prostate has to resort to the catheter following exposure to wet or cold is the day he should have his prostate removed; for then the mortality risk is considerably less than 4 per cent.; the functional result is practically always perfect, and there are no unpleasant complications or sequelæ. This patient was one of those extravagant and stupid persons who summon and pay a doctor, and then discard his expert advice, casting away both money and health at one throw. Such individuals are too unintelligent to appreciate values. The doctor's advice to this patient was worth more than the price of the best automobile on the market; but because the patient bought it at the price of an inner tube, he was unable properly to judge its value.

In place of performing the necessary operation, therefore, the doctor catheterized the patient, and he did it very well. No infection resulted, and the doctor got out of the case with credit to himself. Then the patient thought he could catheterize himself—it seemed so simple. He did so, then had a chill, and an infection followed. Now he has two leaking urinary fistulæ and other sequelæ of infection, and he comes begging that we pull him out of the mire into which he has plunged himself so deeply. We are going to try to do something for him—we can't tell yet how much.

The perineal operation is the one which we shall have to do, using a median incision with a deviation to the left as the rectum is approached, instead of the "U"-shaped incision which we described in 1903. We did a great many operations by the perineal route before we finally turned to the Freyer suprapubic operation, which is now the operation of choice for nine-tenths of our prostatectomies. But one cannot readily get into this man's bladder from above. The extensive suppuration which has occurred in the space of Retzius would make entrance to the bladder difficult and dangerous. Therefore, if this prostate is to be taken out at all, it must be done from below. Even if we do not succeed in removing it in its entirety, we shall at least succeed in substituting a single drainage opening in place of many sinuses.

The next question which occurs to one is, Is this prostatic growth malignant? Cancer of the prostate not infrequently invades the periprostatic tissues, and may produce a clinical picture not unlike the present case. The rule is that a diagnosis of malignancy of the prostate is made from the woody hardness of certain portions of the gland, as compared with the normal consistence of the remainder of it. Here this differential diagnosis cannot readily be made, because the patient has an inflammatory and fibrous infiltration of the entire prostate and its vicinity, and every part of the prostate feels woody. The presence of so extensive a suppuration does not at all rule out a carcinoma, nor does it speak in favor of its presence. A cancerous prostate which has a typical ligneous "feel" before operation may be broken down like a piece of Herkimer cheese by the finger at the time of operation. A fibrous inflammatory prostate, however, is very tough.

We are going into the perineum by a median incision, and then divert the lower half of it to the patient's left. We are going to manage the rectum as best we can, and remove at least a portion of the prostate. This is, at best, a very disagreeable case.

We made a phthalein test on the patient's total urine, and it showed only 35 per cent. in place of 55 per cent. excretion in the first two hours, so you see the kidneys are already below par. His blood-pressure is high and his arterial tension is great. He is an unwelcome surgical risk, but something must be done for him, since his present leaky life is unbearable. These fistulæ must be closed and suppuration stopped. We are going to take the time to do this dissection with the greatest possible care. Formerly I used to like to run my finger around the inside of the capsule and swing out the prostate. The first prostate on which I tried

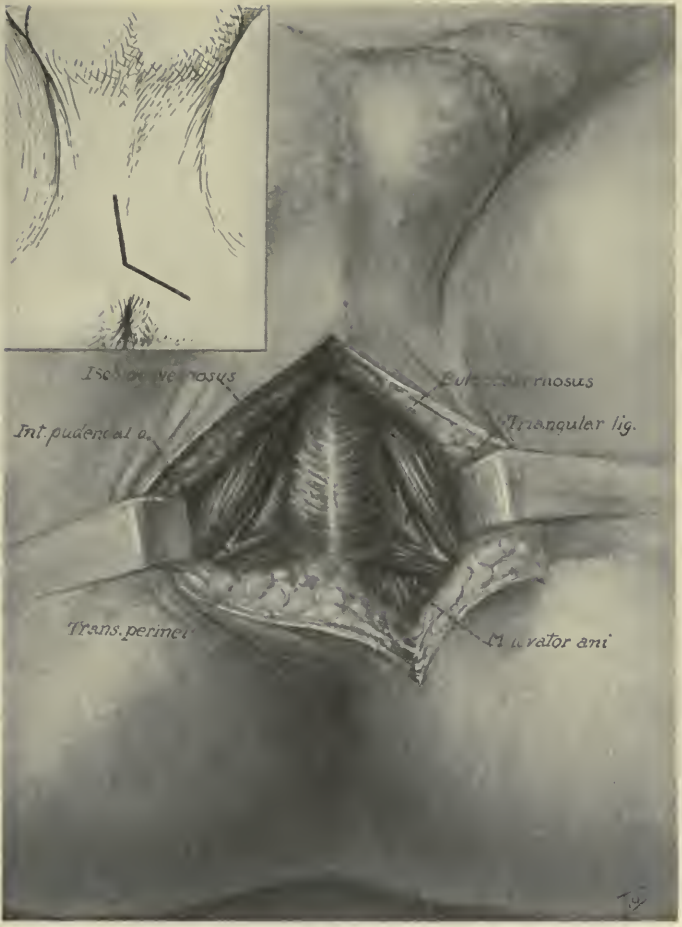


Fig. 80.—Perineal prostatectomy. The insert drawing shows Dr. Murphy's incision in this case, which enabled him to excise the perineal fistula present more easily than his usual semilunar incision would have permitted. Note the full exposure of the field of operation which this atypical incision allowed after the perineal fistula and scar tissue were excised. Compression of the exposed internal pudendal arteries (O. T., internal pudic) reduced hemorrhage to a minimum during the subsequent steps of the operation.

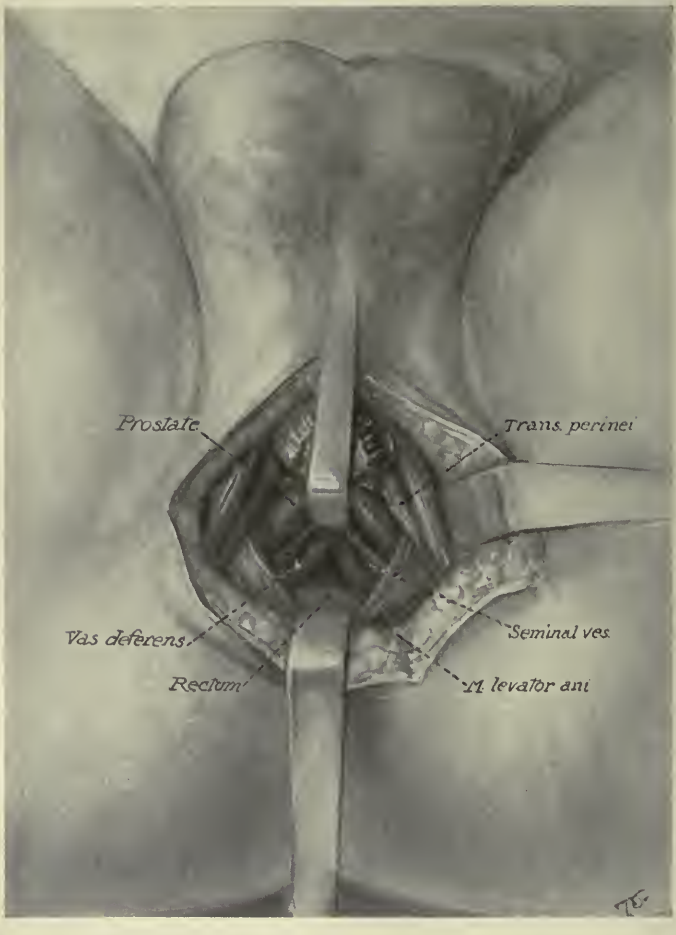


Fig. 81.—Perineal prostatectomy. For the sake of anatomic clearness the structures in the depth of the wound are represented here as lying rather closer to the eye and more distinctly outlined than they actually appeared during the operation, at which they were considerably more obscured by the connective tissue resulting from the long-standing periprostatitis.

this manœuver just popped out, and a visiting doctor sitting here in the clinic in the front row inquired, "Doctor, do they always pop out like that?" That was the only one that ever did come out that way for me by the perineal route. The capsule is so easily separable from the average hypertrophied prostate, as we know from our anatomy, and the prostate is peeled out of it, as a rule, with so great ease, particularly by the suprapubic route, sometimes in a minute and a half, as Freyer very frequently does them, that it seems strange that their removal is so much less easy by the perineal route. Freyer does on an average the best prostate work, I believe, that is done in the world. He can bring out a prostate as easily and quickly as Dr. Joseph Price used to remove pus-tubes. "Joe" Price could go down and bring up a pus-tube, using only his fingers, more rapidly and neatly than any one else I ever knew. The same is the case with Freyer. He runs his finger around the inside of the prostatic capsule, and while we are speaking about the technic he is to use, he has it out. He has it out so promptly because he is able to get at once on the true capsule with his finger-tip and stay on it.

In this case we shall not be able to make out any true capsule, since the prostate and its environment are probably fused into one mass by the inflammatory infiltration. With the suprapubic operation the key to the situation is to get the finger-tip around the catheter and rupture the capsule anteriorly. Then keep working the finger along the catheter until the prostatic urethra is attained. Tear with the finger through the anterior commissure and come at once on the capsule. Enucleate the prostate from the capsule by running the finger around to the right, that is, clockwise. Then the prostate stands out free in the bladder neck except for the urethral attachment at its apex. The urethra is then torn across with the tip of the finger as the hand in the rectum presses the prostate forward. This is the best way to carry out the enucleation. It is the way Freyer does it, and it furnishes the reason why he is able to enucleate the prostate so quickly and so completely.

[Dr. Murphy followed his usual technic in performing this operation with the exception of the incision and save as the connective tissue and pus in the field made the dissection somewhat less typical than usual. The patient was in the lithotomy position, the feet in stirrups, and a thick metal sound in the urethra. After cutting through the subcutaneous fat the wound gaped widely as the cutaneous flap was drawn toward the anus, by the contraction of the anal sphincter. The deep fascia was incised, the tendinous raphé perinealis exposed, together with the adipose tissue filling the ischiorectal fossæ. The muscle-fibers connecting the external anal sphincter with the bulbocavernosus were divided transversely. The bulb was retracted forward, and the transverse perineal muscle, and below it the superficial layer of the triangular ligament, exposed. The triangular ligament was divided close to the anus, and, with the transverse perineal muscle, drawn forward, exposing the compressor urethræ muscle, in which lie the glands of Cowper, and the membranous urethra. The further dissection Dr. Murphy carried on largely with his forefinger-tips, burrowing slowly in, throwing forward the fibers of the compressor urethræ, and spreading the levator ani fibers apart. The pelvic fascia behind the levator ani contained a small abscess and much connective tissue surrounding the prostate and binding it down to the rectum. When this point in the dissection was reached, Dr. Murphy kept one forefinger in the rectum, to protect it, while he dissected with the other. The prostate lay so high up and was so embedded in scar tissue that it was difficult even for Dr. Murphy, with his exceptionally long fingers, to dislodge it. He finally, however, succeeded in getting it away piecemeal.—Ed.]

There is the middle lobe. That is the lobe which did the chief mischief. I could feel it projecting up here inside the bladder for almost half an inch, a teat-like process split down the middle. Here in front there is a pus-pocket which reaches up into the scrotum but has not resulted in perforation.

This is not an ideal prostatectomy by any means. It could not be from the nature of the case. I got the capsule freed quickly enough, but I could not put my finger on all the prostate readily. Now it is all out, however, and we will put the tube in the bladder.

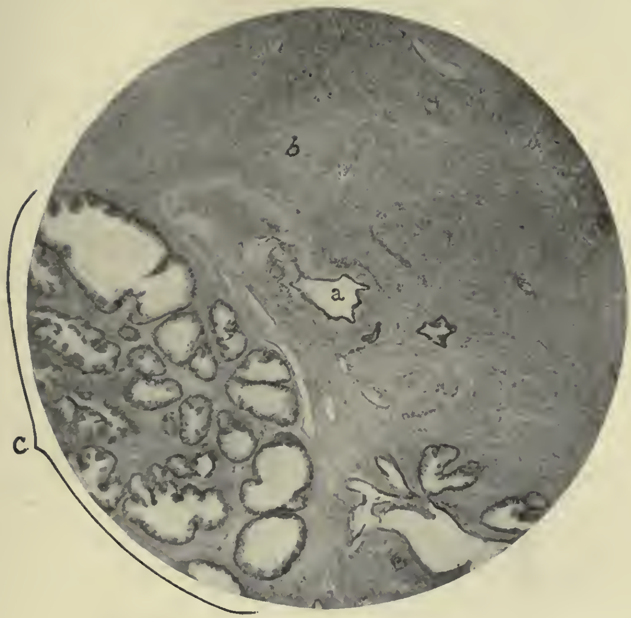


Fig. 82.—Hypertrophy of the prostate. Chronic prostatitis and periprostatitis. The microscopic sections show the normal prostatic tissue (*c*) cut up into islands by great strands of connective tissue (*b*) which course through the gland and occasionally show a few atrophic and compressed remnants of the glandular structures (*a*) embedded in them. This low magnification photomicrograph shows such a mass of connective tissue on the right and a few acini of glands on the left, with connective tissue between them. Note the nearly complete absence of epithelium in (*a*) (preparation by Dr. W. O. Sweek).

Let the record show that there was a band of cicatricial tissue lying below the prostate which constricted the approach to it very materially. This band was finally spread; the prostate was enucleated first from its lateral capsule, while the finger was kept in the rectum all the time, so as to protect the latter from tearing. We have fully protected the rectum from injury, so that its wall is not even thinned. The quantity of blood lost was comparatively small.

That is a very happy outcome of a difficult case. We will irrigate the bladder with warm saline solution, apply a dressing, and return the patient to his room.

VISITING DOCTOR: If you had used a tractor to draw down the prostate, would it not have been easier to reach and peel out?

DR. MURPHY: Perhaps so. But I practically never use Young's tractor or Legueu's *désenclaveur*, because I have exceptionally long fingers, and have always been able to reach and bring down the prostate without these instrumental aids, which are not entirely innocent in their action on the urethra, to which they furnish an additional and, so far as I am concerned, an unnecessary mechanical insult. Likewise, I do not use the Muzeux forceps to seize the gland after it is freed, although I have no doubt all these mechanical aids are of great value to the operator with stubby fingers. I believe that the more I use my fingers in the deep dissection in this region, and the less I rely on instrumental aids, the more likely am I to get out the gland *in toto*, and yet preserve the surrounding structures—the seminal vesicles, vasa deferentia, and the rectum—from injury.

Of course, in a good many cases I presume that our so-called total extirpation is not really total. The "capsule" which we leave probably often contains compressed and atrophic remnants of the gland, just as the capsule of the thyroid usually shows a thin lining of atrophic thyroid tissue after the enucleation of a cystadenoma. I presume that a really total extirpation of the prostate is performed only when the capsule is removed with the gland, and this additional procedure is, therefore, imperative in the treatment of a cancerous gland.

As a rule, in doing the perineal operation I aim to spare the membranous urethra, so far as possible, although in a case like this one of to-day any such effort is practically in vain, the connective-tissue masses blunting all delicacy of technic. I often leave a little of the apex of the prostate rather than tear away the membranous urethra unnecessarily.

After removing the main mass of the prostate I pass my finger in the bladder and search for an enlarged middle lobe, just as I did to-day, and then remove it separately from the rest of the gland. I practically never attempt to draw down the bladder mucosa and unite it to the urethral mucosa, as Zuckerkandl has recommended, because I think this refinement of technic is as unnecessary as it is difficult.

VISITING DOCTOR: How long would you leave in a retention catheter and a tamponade in the average case?

DR. MURPHY: We leave the catheter in long enough to serve as a guide for the formation of a new urethra—from eight to ten days, as a rule. The tamponade we remove in from twenty-four to forty-eight hours and then leave the wound wide open. It is often astonishing how quickly a new urethra forms in these patients and how rapidly some of these distended and apparently atonic bladders regain their expulsive function after the removal of the obstacle to their outflow.

[NOTE (DECEMBER 26, 1914).—The patient made an uninterrupted recovery and did not have a single untoward symptom. There was rapid union of the perineal wound, and all the drainage is now through the urethra. There has been no further discharge of urine through the sinus in the groin since the operation, and the perineal sinus was excised during the operation.—ED.]

SPONTANEOUS MASSIVE COAGULATION OF CEREBROSPINAL FLUID WITH XANTHOCHROMIA.—ITS SIGNIFICANCE IN THE DIAGNOSIS OF LESIONS OF THE SPINAL CORD AND ITS MEMBRANES

A Diagnostic Talk by Dr. Charles Louis Mix

In 1903, in the *Gazette des hôpitaux*, G. Froin, under the title of "Inflammations méningées avec réactions chromatique, fibrineuse et cytologique du liquide céphaló-rachidien," reported three cases observed in Widal's service in the hospital Cochin, in all of which, upon doing a lumbar puncture, a cerebrospinal fluid was obtained which was yellow in color and contained numerous cellular elements, and so large a quantity of fibrin that it coagulated spontaneously. Up to this time no such case had been reported in medical literature, and the cases of Froin's were therefore unique. Lépine, however, had also reported in the same year the case of a patient with tuberculous meningitis whose cerebrospinal fluid showed a remarkable amount of albumin, but no yellow color. Lépine's case, however, does not belong to Froin's group, because the cerebrospinal fluid showed no xanthochromia (yellow color). On account of the general interest of the matter, it may be well briefly to summarize Froin's original cases.

Case i.—The first was a woman, thirty-six years of age, with a right facial paralysis which came on during November, 1902. Beginning April, 1903, a painful flaccid paraplegia set in. Upon her entrance to the hospital four lumbar punctures were made, the first of which was done May 22, 1903. Twelve cubic centimeters of fluid ran slowly into three tubes from the cerebrospinal canal. The cerebrospinal fluid was clear, and in ten minutes had coagulated into a solid mass. After the clot had separated the clear fluid was centrifuged and was found to contain numerous lymphocytes. The second puncture, made June 3, let out 15 c.c. of a slightly yellow cerebrospinal fluid, showing the same spontaneous coagulation and an abundant lymphocytosis. On June 17, 10 c.c. of cerebrospinal fluid were obtained. At first the fluid came out with a slight spurt, but subsequently only drop by drop. It coagulated completely within two hours. This sample was examined carefully for tubercle bacilli and other bacteria. Two guinea-pigs were inoculated with from 2 to 5 c.c. of the fluid. They were killed a month later, but showed nothing pathologic. On August 21, 1903, 12 c.c. of a slightly yellowish cerebrospinal fluid were obtained, which showed a slight coagulum within an hour.

In general the cerebrospinal fluid was never under notable tension, but it was always yellowish. It always contained lymphocytes, and it always either coagulated spontaneously or, as in the case of the last puncture, showed a coagulum after an hour. At the time of the publication of Froin's paper this patient showed the same clinical picture unchanged, which in the light of subsequent cases recorded in the literature seems to have been a pachymeningomyelitis of the conus terminalis with a flaccid paraplegia.

Case 2.—Froin's second case was that of a young woman, twenty-three years of age, with multiple tuberculous foci in her bones. These had begun to manifest themselves when the patient reached the age of eight. On July 16, 1903, after the crest of the left ilium had been cureted, the patient was suddenly taken with fever, restlessness, headache, spinal pains, and showed a slightly positive Kernig's sign. On the sixth day a lumbar puncture was made and only a few drops ran out, because of the fact that during the course of the puncture the cerebrospinal fluid spontaneously coagulated in the puncture needle. After the separation of the clot very numerous polymorphonuclear cells and red blood-cells were found in the fluid which was collected. A culture was made, but no growth resulted. On the twenty-eighth day of the disease the meningitis signs disappeared. Twelve c.c. were again obtained by spinal puncture, but instead of being yellow, as was the fluid at the time of the first puncture, it was clear, and instead of coagulating spontaneously, it showed merely a slender coagulum at the end of an hour. Examination of the centrifuged fluid showed a number of polymorphonuclear cells, but the lymphocytes were especially numerous. In this case only two spinal punctures were made. The yellow color was ephemeral, and the fibrin diminished in quantity very materially during the period between the two punctures.

CASE 3.—The third case reported by Froin was that of a man, forty years of age, who entered the hospital on August 20, 1903, for a paraplegia which had begun on August 14, after he had been chilled. The paraplegia came on very rapidly, and the paralysis spread upward, involving subsequently the muscles of the trunk and the upper limbs. The case ran the classic course of Landry's paralysis, developing without fever. On the seventh day of his illness 12 c.c. of fluid were obtained from him by lumbar puncture. The fluid ran out slowly drop by drop, was of a yellow color, and within half an hour had coagulated completely. Subsequently the clot retracted fairly readily, and in the supernatant serum a large number of lymphocytes were found. The cultures made from the fluid remained sterile. On the ninth day of the illness 16 c.c. of the cerebrospinal fluid were withdrawn, the fluid on this occasion also flowing drop by drop. It was slightly yellowish in color, and coagulated within half an hour. Again the culture was negative. In this case the color of the fluid was not so yellow as in the first two cases, and the lymphocytes were not so numerous. On the other hand, coagulation was more intense than in either of the other two cases.

Froin made no attempts at generalization on the basis of these three cases. He merely called attention to the rarity of the condition, the only instance of which he had ever heard being a case of Ballet's which was reported to him orally by Sicard.

Mestrezat, in his elaborate book on cerebrospinal fluid, published in Paris in 1912, has a chapter devoted to the syndrome characterized by massive coagulation and xanthochromia of the spinal fluid. He calls attention to the fact that if a case is to be placed in this group, it must show spontaneous massive coagulation of the spinal fluid. This clot at times is so solid that the test-tube can be turned upside down without spilling a drop of liquid. In other words, the latter completely gelatinizes. The cases collected by Mestrezat, 21 in number, include those of: Froin (1903), Babinski (1903), Cestan and Ravaut (1904), Ballet and Delherm (1904), Donath (1905), L. Fornaca (1906), Tedeschi (1906), Sicard and Descomps (1908), Froin and Foy (1908), Blanchêtière and Lejonne (1909), Derrien, Mestrezat and Roger (1909), Braillon and Devillers (1909), Claude (1910), Verhoogen and Dustin (1910), Flatau (1911).

The reports above enumerated comprise 18 cases. To these Mestrezat adds 3 hitherto unpublished cases, making a total of 21. Mestrezat, in his book, considers, first, the clinical syndrome, together with a summary of the 21 cases, and then the chemical and humoral syndrome. After that he discusses the pathogenesis and the diagnostic, prognostic, and therapeutic indications.

Aubrey, in his *Thèse de Paris* in 1909, writing on the subject of massive coagulation of the cerebrospinal fluid, collected, in all, 12 cases up to 1909. These cases Mestrezat summarizes, and to them adds 9 later ones.

Case 4.—This case, reported by Froin, appears only in the *Thèse de Paris* of Aubrey. The patient showed multiple lesions of the spinal cord, probably of syphilitic origin. There was optic atrophy and paralysis of the external rectus oculi and an Argyll-Robertson pupil. The symptoms in the lower extremities were especially marked. The pains first appeared in the left leg and then in the right. Subsequently there was an atrophy of the left leg, with loss of the spinal reflexes and a slightly positive Kernig's sign. The diagnosis by elimination wavered between a multiple sclerosis and a syphilitic cerebrospinal meningitis. A lumbar puncture was made, and the characteristic yellow fluid obtained and found to contain lymphocytes. The case is not well reported, and the localization of the process was evidently not clean cut. From the point of view of disclosing the pathogenesis of the cerebrospinal fluid obtained this fourth case of Froin's is worthless, notwithstanding that it did show massive coagulation and xanthochromia.

Case 5.—Babinski's case, the fifth on record, was reported in 1904. It was a spastic paraplegia. On account of the contractures, exaggerated reflexes, the Babinski toe-sign, and the sphincter disturbances, the diagnosis of pachymeningitis was deemed likely. Sensation was only slightly disturbed, however. Lumbar puncture disclosed the spinal fluid characteristic of this syndrome. Under vigorous antisyphilitic treatment with mercurial inunctions there was a slight improvement noted in the patient's condition for a period of eight months. The lumbar puncture itself also seemed to exercise a favorable influence. As nearly as can be determined from the report this case falls very well into the category of spinal compression caused by a fibrinous and hemorrhagic pachymeningitis, as diagnosed by Babinski, with probably a pocketing of the fluid in the lower portion of the spinal canal.

Case 6.—In 1904 Cestan and Ravaut reported a case of what they called pachymeningomyelitis of the conus terminalis with postmortem examination. In this case the cerebrospinal fluid showed the yellow color, or xanthochromia, and coagulation *en masse*, with the presence of an increased number of cellular elements. By the time they had written their article, six cases of this sort were on record, three more having been added to the three reported by Froin. These authors, therefore, reported the seventh case. Two factors were thought by them to figure in the explanation of this curious spinal fluid syndrome of xanthochromia, coagulation *en masse*, and the presence of numerous cellular elements. These two factors were, first, meningeal inflammation, and, second, hemorrhage. Thus the authors explain the yellow color and the huge amount of albumin present: the deep yellow color resulting from the dissolution of red blood-corpuscles and consequent freeing of the hemoglobin; and the enormous amount of albumin being due to serofibrinous exudate of the inflammation. The two factors may coexist in a case, and one may, therefore, venture upon the diagnosis of a fibrinous hemorrhagic meningitis, as Babinski did in a case which he reported immediately after Froin's original publication. Cestan and Ravaut were of the opinion that, during the active stage of the meningomyelitis, a hemorrhage had probably taken place.

Cestan and Ravaut's case resulted fatally, and was the first case ever to come to postmortem examination. At the autopsy these authors found a fibrous concrescence of the meninges and cord at the level of the conus terminalis, this concrescence being caused by the pachymeningitis, which was annular in its development.

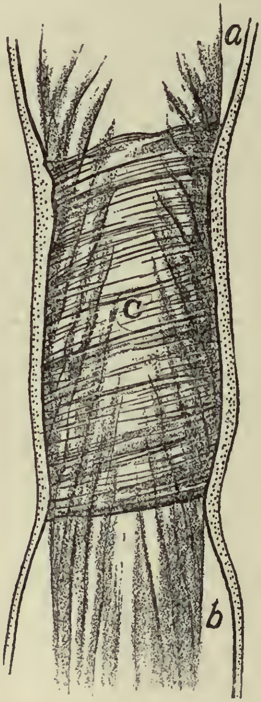


Fig. 83.—Chronic meningomyelitis involving the conus and upper caudal filaments (*c*) and obliterating the meningeal space over the area involved, thus isolating the lower meningeal sac (*b*) from the upper (*a*) and blocking the circulation of the spinal fluid. It is in the isolated lower sac that the spinal fluid presents the syndrome of xanthochromia, coagulation *en masse*, and lymphocytosis. Dr. Murphy, for the relief of this condition, has proposed an operation consisting of excision of the membrane, and the dura mater, too, if adherent. The dural defect is then filled by implantation of a pedicled flap of fascia or muscle (after Cestan and Ravaut).

Case 7.—Ballet's case, included by both Aubrey and Mestrezat in their collection of cases, was based wholly upon an oral communication to Cestan and Ravaut of the existence of such a case, and is merely reported in a foot-note in their paper. It has no value in explaining the origin of the syndrome.

Case 8.—The case by Donath (1905) was one which clinically showed a Landry's syndrome. There was an ascending paralysis, beginning in the lower extremities and reaching ultimately the upper extremities and the face, with bulbar disturbances. The reflexes were lost. There were disturbances of sensation in the lower extremities and a loss of sphincter control. Subsequently these symptoms all faded away after a duration of three months. The case ultimately recovered completely. Reading over the report of the case leaves one with the impression that there was never any accurate topographic localization of the disturbances. The cerebrospinal fluid, however, showed massive coagulation and xanthochromia.

Case 9.—The case of L. Fornaca was published as an example of the conus terminalis syndrome. There was saddle-shaped anesthesia, with lost knee-jerks and disturbance of the sphincters. Moreover, there were trophic disturbances in the feet, muscular atrophy of the legs, and decubitus. There was no typical Kernig's sign elicited, though there was a suggestion of it. There was severe pain in the back, radiating into the lower extremities. The patient left the hospital not yet recovered after remaining there for a month. Perusal of the report leads one to infer that the case was one of meningomyelitis, with a definite localization in the conus terminalis. It undoubtedly belongs to that group of cases in which a pocket formation of the conus terminalis is secondary to pachymeningitis.

Case 10.—This case was reported by Tedeschi in 1906, and showed a paraplegia, the paralysis being more pronounced in the left leg than in the right. It produced the usual syndrome of the conus terminalis, with lost knee-jerks and sphincteric disturbances. There was such violent pain in the sacral and coccygeal region that it necessitated the removal of the coccyx. There was the characteristic saddle-shaped anesthesia. This patient died. The postmortem examination showed a swelling at the level of the conus terminalis or slightly above it. There was a growing together of the meninges and spinal cord which was quite extensive, particularly on the left side. The inflammatory growth was organically fastened to the inner surface of the dura mater. It also enveloped the pia arachnoid of the cord and the conus, as well as the nerve-roots, having begun either at this level or having come from a point higher up. Microscopic examination showed a thickening of the meninges without any pachymeningitis externa. The vessels also showed thickened walls. Some were even obliterated. Tedeschi states that the etiology of the case could not be worked out. It is evident, from a perusal of his case, that there was a complete isolation of the lumbar culdesac.

Case 11.—The next important addition to the literature was contributed by Sicard and Descomps in the *Gazette des hôpitaux* for October 20, 1908. These authors began their report by stating that the syndrome of xanthochromia and coagulation *en masse*, with the presence of numerous cellular elements and a distinct lymphocytosis, up to that time had always been associated with paraplegia, and that it had always been caused apparently by an active meningomyelitis with local and circumscribed edemas. The case which they then reported, taken from Brissaud's service, was that of a woman, thirty-nine years of age, not of alcoholic tendencies, admitted to the ward in January, 1908. She was suffering from a paraplegia which had begun painlessly fifteen months previously and had steadily progressed. For several weeks she had been confined to bed. The paraplegia, instead of being flaccid, in her case was spastic, with diarrhea, contractures, and tendinous retractions. There were also disturbances of the sphincters, trophic sores, decubitus, and both subjective and objective sensory disturbances. No changes were found in the spinal column.

Four spinal punctures were made, all of them between the fourth and fifth lumbar vertebræ. The first, made February 5, 1908, showed a yellow fluid which coagulated almost as soon as it began to flow, so that only 4 c.c. were obtained. From the clots in the test-tube some serum subsequently separated, which, when centrifuged, showed red blood-cells and numerous lymphocytes. The clot, which was examined, also contained red blood-cells and many lymphocytes. A second puncture, performed on February 11, curiously enough gave a perfectly limpid cerebrospinal fluid without any spontaneous coagulation, but with fairly numerous lymphocytes, nevertheless. On February 26 lumbar puncture again showed a yellow fluid, with spontaneous coagulation and lymphocytosis. Two days later, on February 28, the cerebrospinal fluid was once more limpid, did not coagulate, and showed only a moderate lymphocytosis. This case was interesting by reason of the variations from time to time in the character of the fluid obtained by lumbar puncture.

At the postmortem examination, which was performed during the middle of March, 1908, a grayish-white mass was found which had involved the pia-dural space at the level of the eleventh and twelfth dorsal vertebræ. It extended somewhat above and somewhat below this level and was adherent to the dura. The meninges were thickened, so that there was a uniform and true perimedullary sheath from the tenth dorsal to the second sacral segment of the cord. This sheath also enveloped the nerve-roots. In places there was an agglutination between the dura and the pia encircling the cord like a sheath, and made up of very dense fibrous tissue. In places the pia was elevated above the surface of the cord by patches of edema from 2 to 5 cm. in extent, which looked like blisters of the cord. In this area the blood-vessels were much engorged. On microscopic examination of this grayish-white, sclerocaseous area a number of giant-cells were found. The process was believed to be bacterial in origin; but no tubercle bacilli could be stained in the tissues and no inoculation of the tissues was made into guinea-pigs; hence the etiology is still in doubt. This case reminds one of Froin's second case.

Sicard and Descomps rejected Babinski's suggestion that the xanthochromia, coagulation *en masse*, and lymphocytosis were caused by a rather diffuse hemorrhagic fibrinous meningitis. These authors believed rather that the inflammation was annular in its distribution, so that it caused vascular compression and, subsequently, local edema in the meningeal pouch which forms beneath the level of the constriction. These authors apparently were the first to recognize the fact that the essential factor in the production of xanthochromia with coagulation *en masse* is the formation of a meningeal pouch. Sicard and Descomps thought that plasma exuded into this meningeal pouch. They thought that the pouch in their case was formed by very slight adhesions, because at times limpid cerebrospinal fluid trickled into it from above. Thus they explained the variability in the fluid which they obtained at different spinal punctures and the surprisingly rapid appearance and disappearance of the yellow color of the fluid.

These authors conclude their article by stating that, on the whole, the syndrome is of diagnostic value, always indicating a meningitis and never a tumor. Indeed, they go so far as to say: "Supposing that the etiology in a case of paraplegia be undecided and that the diagnosis rests between a meningomyelitis and a compression of the cord by a tumor? If the lumbar puncture reveals the syndrome of xanthochromia, coagulation *en masse*, and hematolymphocytosis, one might reasonably infer that the disturbance is not due to a tumor (fibroma, sarcoma, psammoma, cyst, etc.), but to a meningomyelitis, probably either tuberculous or syphilitic, and extending over quite a stretch of the meninges. This would be an interesting and important diagnostic point if the idea of operation were entertained." As we shall subsequently see, this conclusion of Sicard and Descomps was subsequently proved to be false by the report of further cases, including our own.

Case 12.—Froin and Foy. This case was one of cerebrospinal meningitis consequent upon an intraspinal injection of stovain, the cerebrospinal meningitis appearing very shortly after the injection. There was a very marked Kernig sign and severe pain in the lower extremities. It is not likely that the trouble was due, as is implied, wholly to the stovain, because the stovain was used that an injection of collargol might be made. The striking point about this case is the demonstration of the non-absorption of the collargol which was injected. The case, when analyzed, points to a meningeal process localized fairly sharply at the level of the lumbar cord. A perusal of the case report indicates that either an agglutinating process or, possibly, a constricting one, was the pathologic change present. This case also is suggestive of the formation of a lumbar culdesac.

CASE 13.—The next important case of xanthochromia, coagulation *en masse*, and lymphocytosis was that of Blanchêtière and Lejonne, reported in the *Gazette des hôpitaux* for September 14, 1909. These authors reported their case rather thoroughly. It was a spastic paraplegia due to a sarcoma of the spinal dura mater in the dorsal region, without any appreciable accompanying involvement of the arachnoid and pia. The patient, a male, sixty-six years of age, entered Raymond's service at the Salpêtrière May 8, 1907, because of his spastic paraplegia. The onset was in January, 1907, with cramps and weakness of the right leg. Two weeks later the same symptoms appeared in the left leg, so that the patient was thereafter compelled to walk with crutches. Later a complete paraplegia gradually developed· Examination on May 20, 1907, showed a spastic paraplegia with sensory defects up to the level of the seventh dorsal vertebra. His legs were completely paraplegic with the exception of some slight feeble movements. The Achilles and knee-jerks were very lively, and there was a patellar clonus. The Babinski toe-sign and the Oppenheim sign were both positive. The cremasteric and abdominal reflexes were absent. Sensation was pronouncedly disturbed in the lower half of the body. Sensation to touch was much diminished, and the patient made serious mistakes in localization in his legs and trunk up to the level of the ninth dorsal segment. Pain and temperature sense were absolutely gone as high as the tenth dorsal segment. Just above this segment there was a zone of hyperesthesia in the territory of the ninth and eighth dorsal segments. Muscle and joint sense were also lost. There was some disturbance of the bladder sphincter, but the patient knew when his bladder was full, though he had difficulty in expelling its contents. There were no visceral symptoms of any sort. There were no pains along the spinal column, even when the spinous processes were struck a rather sharp blow.

A lumbar puncture was made May 22, 1907, and a specimen of yellow fluid was obtained which coagulated *en masse;* but only a *very few* lymphocytes were found when the separated serum was centrifuged. The diagnosis was very easy as to localization, but very difficult as to etiology. It was still further complicated by the result of the lumbar puncture, because of the negative cytologic findings in the presence of xanthochromia and coagulation *en masse.* The history was entirely against the lesion being a meningitis. Three possibilities presented themselves. First: the prominence of certain spinous processes suggested a Pott's disease; but there was no local pain or tenderness in the spinal column and no signs of tuberculous infection elsewhere. Second: the diagnosis of tumor of the cord or its membranes was possible; but, curiously enough, there was no pain, and pain has always been regarded as a constant initial sign of spinal cord tumor. Third, a transverse myelitis of undetermined etiology had to be taken into consideration.

The second lumbar puncture was made May 28, 1907. The fluid withdrawn was exactly like that removed May 22. On June 18 the condition of the patient was the same as at the previous examination, except that the spasticity had increased and painful cramps were setting in in the lower extremities. The third lumbar puncture gave precisely the same sort of fluid as the second. The fourth puncture was made July 5, and a guinea-pig was inoculated with it. This pig was killed July 26 and showed absolutely no evidence of tuberculosis. On October 8 lumbar puncture gave issue to a fluid which showed unchanged findings and coagulated almost at once. The punctures done October 27, 1907, and January 6, 1908, gave the same results.

About the beginning of 1908 the patient began to show a certain amount of muscular atrophy, especially in the left thigh, and the sphincters of the bladder became involved. The rectal anesthesia had increased to such a degree that the patient could not feel fecal matter pass. Between January, 1908, and September, 1908, the patient's condition continued to deteriorate. Lumbar punctures done September 24 and September 30, and again November 21, showed the same yellow color with coagulation *en masse.* A spinal injection of magnesium sulphate, made for the purpose of overcoming the contractures and the intense pain in the legs, brought with it no lasting results, though for forty-eight hours there was some relief. Death finally occurred in March, 1909, a little over two years after the onset of the trouble.

The postmortem examination was made thirty hours after death, the body previously having been embalmed with formalin. There is no explanation for the delayed postmortem examination offered in the article quoted. The cerebrospinal fluid at the time of necropsy was very light yellow in color, and contained less fibrin and albumin than that removed from the body before death. A great many cells, however, were found in the fluid, chiefly red blood-cells, polymorphonuclear cells, and some large cells with several nuclei, staining badly. This finding was unlike the condition present during life, in which the cellular elements in the cerebrospinal fluid were extremely sparse. The brain was absolutely healthy, and the spinal column was free from any tuberculous lesion.

A tumor of the dura mater of the form and dimensions of a large olive was found situated on the dorsal surface of the cord, which it compressed at the level of the seventh, eighth, and ninth dorsal segments. It appeared to be somewhat adherent to the cord, but with slight traction was easily separated from the latter. There was no trace of meningitis, but three or four secondary foci in the pia, each a little larger than a pin-head, were found on the dorsal surface of the cord and a little below the main tumor. Microscopic examination showed the tumor to be a small, round-cell sarcoma, containing numerous vessels engorged with blood and multiple hemorrhagic foci. The spinal cord was distinctly compressed. There was no sign of a meningitis.

During the course of the patient's illness seven punctures in all were made, and they all showed the same characteristics. In other words, there was no alternation between a limpid cerebrospinal fluid without coagulation and the syndrome of xanthochromia, coagulation *en masse*, and hematolymphocytosis. In this case the cerebrospinal fluid sometimes coagulated in the needle so that a wire had to be used to get it to flow. The clot, after it had coagulated, easily retracted and the residual fluid was collected. This was centrifuged and showed only a few large cells,—never more than three or four lymphocytes to the field,—and no red blood-cells were found.

These authors were the first to analyze the cerebrospinal fluid, and since they are the only authors who have done so, except Mestrezat and Roger, their results are quoted.

ANALYSIS EXPRESSED IN PARTS PER THOUSAND

	October 27, 1908	September 24, 1908	September 30, 1908
Density	..	..	1015
Dry extract	..	..	47
Chlorids	..	7.39	7.55
Urea	..	..	.59
Fibrin	1.70	1.61	1.63
Globulin	8.08	27.25	27.66
True albumin	17.42		

Spectroscopic examination showed no hemoglobin.

These authors pointed out that the cerebrospinal fluid which they obtained from their case was unlike the other cases in two respects: first, it was constantly the same; second, it contained no cells except three or four lymphocytes to the field. Previous authors had noticed a variability in the cerebrospinal fluid with respect to the disappearance and reappearance of the yellow color. These authors also studied the mechanism of coagulation and found it to be exactly like that of the blood,

Blanchêtière and Lejonne criticize the diagnostic law laid down by Sicard and Descomps by calling attention to the fact that such cerebrospinal fluid did not necessarily mean meningomyelitis, since it could occur in cases of tumor, as their case proved.

As to pathogenesis, the authors reviewed Cestan and Ravaut's idea that it resulted from hemorrhage complicating a focus of active meningomyelitis, and also the hypothesis of Sicard and Descomps, who thought the fluid to be the result of meningeal inflammation circumscribed by an agglutinating process, with vascular compression and secondary local edema. Their tumor case, of course, makes the explanation of Sicard and Descomps valueless, yet they incline to the view that the xanthochromia is derived from hemoglobin. To account for the yellow color they state that the tumor itself showed hemorrhages within it, and they venture the hypothesis of possible small and almost continuous hemorrhages over a period of sixteen months from the tumor. Very important in the light of our subsequent knowledge is the conclusion which these authors arrived at—"Besides the syndrome Froin met with in certain flaccid and spastic paraplegias, having for origin a meningomyelitis, there is another syndrome observed by us for the first time, likewise characterized by xanthochromia and coagulation *en masse*, but without hematolymphocytosis." The symptomatology is the same, but it is due to a tumor of the membranes of the cord. They close by admitting: "It is probable that in our case an early operation would have saved the patient."

CASE 14.—This was reported by Derrien, Mestrezat and Roger, in June, 1909, in the Société de Biologie. The patient was a vineyard laborer, twenty-nine years of age. He entered the hospital March 12, 1909, for a paraplegia. The onset was in June, 1908, with fever and weakness and pain in the right leg. Then the symptoms appeared in the left leg. In December, 1908, because of the contractures which had come on in the right leg, he took to his bed. At the first examination, made in March, 1909, the paralysis was almost complete on the right side, but on the left the patient could slightly raise his leg above the level of the bed. He could only feebly execute movements. Moreover, when he tried to move, contractures often appeared. He was able to take only a few short steps when helped by two people.

On the right side there were disturbances of sensation, hypesthesia to touch and pain, and delayed sensation. Sphincteric disturbances were also present. The knee-jerks were markedly increased, and there was a double patellar and ankle-clonus. The cutaneous reflexes were absent. There was pain on deep pressure over the sacrolumbar muscles on the right side, at the level of the lumbar vertebræ. There was no history of syphilis or tuberculosis.

May 6, 1909, the patient was examined again. The paralysis of the left leg since the previous examination had increased and receded, so that it was not quite so severe as it had been a few weeks back. On the right side the amelioration was notable. The patient moved his leg almost normally. Walking was possible with two canes. The reflexes were still increased on both sides, but there was no patellar or ankle-clonus. The sphincter was in good control. In July, 1909, the patient was found to be much the same as in May. September 4 he left the hospital without further improvement.

About a dozen lumbar punctures were made while the patient was in the hospital. Nine were especially investigated. From the beginning the cerebrospinal fluid was yellow and coagulated, either spontaneously into jelly or after the addition of a few drops of blood-serum. The coagulation and xanthochromia diminished speedily from March until the first few days in April, when the coagulation syndrome began to increase again to the maximum, which it maintained during the rest of his stay. Cytologic examination, obtained by centrifuging the coagulum, showed on many occasions hematolymphocytosis.

This patient's course was followed after he left the hospital. In March, 1910, the patient was not much better than he was in July, 1909. The liquid on this occasion coagulated in the needle at the time of puncture. In December, 1910, the patient showed marked improvement. During the middle of August, 1910, he began to walk a little and the pains in the lower extremities became less violent. By December he was able to walk about five kilometers, and was working a little. In March, 1911, he was back at work. His gait was not spastic, and his strength was almost normal. A bilateral Babinski, however, was still present. In May, 1911, he still had some slight urinary disturbances and was at work in the vineyard. He reported that he did not get tired even at night. The case was evidently one of meningomyelitis clearly localized in the lower part of the cord, and very clearly causing a pocketing of the lumbar culdesac.

Case 15.—Claude. This was the case of a young boy with flaccid paraplegia from a meningomyelitis, also associated with a marked alcoholic polyneuritis. Symptomatically it showed a flaccid paraplegia with lost knee-jerks and the usual meningeal syndrome. There was both constipation and vomiting. There was well-marked stiffening of the neck. Clinically, the case belongs to the group of meningomyelitis of the conus terminalis, and indicates a pocketing of the lumbar culdesac. It was complicated with an alcoholic multiple neuritis.

CASE 16.—Braillon and Devillers. This case, briefly reported, undoubtedly belongs to the syndrome of massive coagulation and xanthochromia of Landry's type.

CASE 17.—Verhoogen and Dustin's case was one of pseudoparalysis. There were paretic and painful disturbances, beginning in the upper extremity and spreading to the lower. Postmortem examination showed a tuberculous caries of the fourth cervical vertebra. The pus which came from the caries caused a compression of the dorsal cord, the fibrinous and purulent material enveloping all the dorsolumbar cord. In this case the blocking-off of the lower portion of the cerebrospinal canal from the upper was quite evident.

CASE 18.—Flatau. This case was reported in the Proceedings of the Neurological and Psychiatric Society of Warsaw, November 19, 1909, and only such brief reviews as appear in French journals are accessible to those not reading Polish. It probably was a case of meningeal sarcomatosis with compression of the spinal cord. Inasmuch as the case is incompletely reported in the French journals, it is not possible to come to an accurate conclusion. There was a paresis of the lower extremities with lost Achilles jerks, a saddle-shaped anesthesia, and some urinary disturbances.

By the year 1912 the subject matter of this syndrome had so increased that Mestrezat, in his book entitled, "Liquide céphalorachidien normal et pathologique," published in Paris in 1912, collected 21 cases, namely, the 18 above reported and 3 original observations. His search of the literature apparently was very extensive. Mestrezat's three cases follow:

CASE 19.—This case, reported only in Mestrezat's book, which is hard to obtain, may on that account be summarized. It was a case of spastic paraplegia with subjective pains in the lumbar region and the lower extremities. There were, moreover, gastric disturbances. The patient was a male, forty-eight years of age, who entered the hospital in May, 1910. The onset of his trouble was ten months previously with dyspepsia. Two months previously he began to suffer from a complete loss of power in his legs and from pains and sphincteric disturbances. Examination showed a spastic paraplegia predominating on the right side. His knee-jerks were increased. There was a double ankle-clonus and a double Babinski toe-sign. Pain was present in the lumbar level and in the lower extremities. There was some loss of sensation, particularly in the right lower limb. The diagnosis of a meningomyelitis, probably from a neoplasm secondary to a gastric carcinoma, was made and a lumbar puncture was done, disclosing the characteristic fluid of this syndrome. The diagnosis was, however, wrong, for on February 26, 1911, his digestive disturbances disappeared. His knee-jerks and spinal reflexes were still increased. There was, however, no incontinence. He was able to walk with two crutches. In May, 1911, the patient began to resume his occupation. This case apparently belongs in the group of meningomyelitis blockading the conus terminalis.

Case 20.—The patient, a male, twenty-three years of age, entered the hospital in December, 1909, with pains in the left knee in front and in the left thigh. These had come on five months previously after his military service. The pains were very severe and kept him from sleeping. He entered the hospital with the diagnosis of a coxalgia and hysteria on account of his fanciful story and the queer course of his case. The diagnosis of hysteria seemed borne out by the absence of the pharyngeal and corneal reflexes. His left knee-jerk was very weak. His anterior crural nerve on the left side was very painful to pressure. There was a diminished electric excitability, so that the diagnosis of a neuritis of the anterior crural nerve seemed tenable. A lumbar puncture was then done, and the liquid found to show a marked xanthochromia with massive coagulation by the next day. Subsequently two other punctures were made, which improved his condition, but an isotonic injection of sodic salicylate brought on the syndrome of the conus terminalis, with saddle-shaped anesthesia, retention of urine, constipation and incontinence of the feces, and later an inflammation of the rectal and urethral mucous membranes. These signs continued for a month. In May, 1910, he was much improved and began to walk again. By December, 1909, he was practically well, except for a slight trouble in urinating when standing. In May, 1911, he was back at his work without the saddle-shaped anesthesia, with normal knee-jerks and with muscular strength unimpaired. This case undoubtedly belongs to the category of an irritative meningomyelitis from the injection of sodic salicylate with a walling-off of the lumbar culdesac.

CASE 21.—This patient was a woman, fifty-eight years of age. In general she showed a flaccid paresis of the lower extremities, with sudden onset, with pain in the lumbar region. The reflexes were preserved, though there were sphincteric disturbances, and later on sensory losses and trophic changes also appeared. A clinical diagnosis of a spinal cord tumor at the level of the eighth and ninth dorsal vertebræ was made.

She entered the hospital October 23, 1910, for a pain in the left lumbar region which she had had for a month. She also had a slight fever. In a few days these went away, but returned the next month on the eleventh, when she was suddenly seized with a paraplegia. There was also trouble when she urinated. There was pain in the spine and pain in the right lumbar region, but none in the lower extremities; nevertheless, there was a feeling of formication. Examination at that time showed a complete flaccid paralysis, with normal sensation and preserved knee-jerks, but there was pain on pressure over the lumbar region. Sensation was present at the level of the eighth and ninth dorsal vertebræ on palpation. It was further thought that the vertebral column was weakened by a neoplasm at this point and that the trouble was secondary to this.

A lumbar puncture was done on November 18, 1911. The coagulum was very slight, but it showed that lymphocytes were present and also altered red blood-cells. On November 23 it showed massive coagulation and xanthochromia. On this date there were ankle-clonus, trophic ulceration, and also a retention of urine. The cytologic diagnosis on this occasion showed large mononuclears with some cells with karyokinetic figures.

With the diagnosis of a malignant tumor of the spine the patient went to the surgical department. The hyperesthesia spread to the left lower extremity and upward to the navel. There were also girdle pains. A sacral ulcer also formed, giving rise to a hectic fever and subsequent death.

The autopsy, which was made eighteen hours after death, was reported by Mestrezat. It is perhaps sufficient to say that there was a meningospinal infection with extradural inflammatory neoformation; that there was a pachymeningitis externa, radiculitis, and an acute poliomyelitis from bacterial emboli. The dura mater on its outer surface seemed to be normal when the spinal cord was opened. At the level of the eighth and ninth dorsal vertebræ, however, the bony canal was narrowed considerably, being in juxtaposition with the meninges, but there were no adhesions externally on the posterior surface of the dura. The spinal vessels at this point were very much congested. On the other hand, the anterior surface of the dura mater showed important changes. At the level of the body of the ninth and over a part of the eighth dorsal vertebræ there were very dense adhesions in which the meninges had to be grubbed away from the bone, and the cord and its meninges detached. The adhesions were found to have been occasioned by a reddish mass, 5 to 6 mm. thick, of fleshy appearance, but of rather soft consistence. This mass adhered to the dura along a stretch of 6 cm., and at the level of the ninth dorsal vertebra had made a kind of sac, which had closed off the end of the cord. Sections of the membranes showed the cord to be very anemic at that level. One gathers from this case that the inflammatory process had very clearly cut off the lower portion of the cerebrospinal canal from the upper.

Mestrezat's collection of 21 cases seems to have been made without thorough study of the German literature. It is true that but very few articles have appeared in German periodicals upon the subject of xanthochromia with spontaneous massive coagulation of the cerebrospinal fluid, yet cases have been reported, and they are here added to the list of known cases.

Important is a paper by Wilhelm Raven, in the *Deutsche Zeitschrift für Nervenheilkunde*, 1912, vol. xliv, pp. 380–416, entitled, "Die Bedeutung der isolierten Eiweissvermehrung und der Xantochromie im Liquor cerebro-spinalis für die Diagnose von Compression des Rückenmarks; Erfahrungen an 15 durch Operation oder Sektion diagnostisch sichergestellten Fällen." Case 12 of this list was a case of Pott's disease. Below the level of involvement the fluid obtained was clear yellow. Raven, evidently, however, was not acquainted with the French literature, for he failed to report any of the French cases. He has collected altogether 47 cases of yellow cerebrospinal fluid, but none of the cases showed coagulation *en masse*. Raven's 47 cases, therefore, do not belong to this syndrome, but merely show a cerebrospinal fluid stained by hemoglobin derivatives from old hemorrhages.

An article by Joseph Reich, entitled, "Ueber Gelbfärbung der Cerebro-spinalflüssigkeit," published in the *Mittheilungen a. d. Grenzgebieten der Medizin u. Chirurgie*, 1912–1913, vol. xxv, p. 721, also has no reference to spontaneous coagulation *en masse*. An article by Edward Schwartz, published in the *Deutsche Zeitschrift für Chirurgie*, 1913, vol. xiv, p. 96, entitled, "Über Xantochromie des Liquor cerebrospinalis," also contains no reference to massive coagulation. The only cases which I have been able to find in German literature are four, in an article entitled, "Ein eigenthümlicher Liquorbefund bei Rückenmarkstumoren (Xantochromie, Fibrin-gerinnung, und Zellvermehrung der Cerebrospinal Flüssigkeit," published in the *Monatsschrift für Psychiatrie und Neurologie*, 1910, vol. xxviii, p. 346, by Otto L. Klieneberger).

CASE 22.—The first of these undoubtedly belongs in the group of spontaneous massive coagulation and xanthochromia. The patient was taken February 24, 1908, after pneumonia, with a very painful paraplegia in the soles of both feet. This passed away, and he was in good health for two years. About six months before he appeared at the clinic he began to experience a peculiar feeling in his hips and back. Following this there were extraordinarily violent attacks of pain in the hips, back, and coccyx. These pains lasted four weeks and then disappeared, to return two months later with redoubled violence. They were always made worse when his body was shaken, and were extreme when he coughed or sneezed. Moreover, coughing or sneezing brought about a peculiar feeling in the back, as though a weight were there. During the following month an equally progressive paresis of both the lower extremities appeared. Examination made by Klieneberger showed a marked paresis of all the muscles of the lower extremities, including the glutei and the iliopsoas. On standing there was a slight lordosis of the lumbar vertebræ. Bending the trunk brought about a spasticity of the erector spinæ. There was no atrophy of the leg muscles, but they were strikingly flaccid. Passive mobility was normal. There was no pain on pressure over the muscles and over the great nerve-trunks. The knee- and ankle-jerks were absent. His epigastric and abdominal reflexes were present. The left plantar and left cremasteric reflexes were weaker than the right. Electric response of the leg muscles was unchanged, although there was a slight quantitative reduction. The vertebræ were not sensitive to pressure. There were no sensory disturbances and no bladder or rectal incontinence. The *x*-ray plate of the spine was negative, with the exception of a lighting up of the bone shadow over the bodies of the second and third lumbar vertebræ.

Two lumbar punctures were made within the course of a week, the first between the third and fourth lumbar vertebræ and the second between the second and third. The fluid escaped under strong pressure. In color it was a clear amber with much fibrin. Very shortly a clot formed and was removed. In a short while another clot appeared. Examination showed an increase of the lymphocytes. There were no other cells. The specific gravity was 1014. The fluid contained 3 per cent. of albumin. Blood-pigment was not discernible.

Klieneberger did not think the cerebrospinal fluid which he obtained was true cerebrospinal fluid. To him it looked as if he had tapped a cystic tumor, although he entertained the possibility of a pachymeningitis hypertrophica. This patient was operated on the twentieth day of March, 1908, by Küttner. A tumor which had grown to a portion of the fibers of the cauda equina and also to the anterior dural wall was removed. It was very soft and about the size of an ordinary date. It lay between the second and third lumbar vertebræ. At the operation he observed below the tumor a clear yellow fluid under pressure. The fluid shot out when the dura was opened. Above the tumor the cerebrospinal fluid was also under pressure, but was clear.

After the operation both Klieneberger and Küttner were apparently unable to decide whether the fluid came from a cyst. They were inclined to the view that the fluid was not cystic. At the same time they recognized that it was not like the normal cerebrospinal fluid which they found above the tumor. At the time of this operation Klieneberger was aware of only two similar cases—the one reported by Tedeschi, "Syndrome della coda equina," in the *Gazz. d. osped.*, vol. xxvii (Case 10), and the case of Cestan and Ravaut, which was the sixth one on record.

Case 23.—The second case reported by Klieneberger also belongs to this syndrome. The patient entered the hospital January 24, 1910, and left February 1 of the same year. When she was twenty-four years of age the patient had gall-stones. At the time of her entrance into the hospital she was twenty-eight. The last attack of gall-stones occurred in the summer of 1908. On the fifth day of August, 1908, she was suddenly seized with violent aching pains in the back and along the outer side of the left knee and in the middle of the calf. These increased for two weeks and were very violent. Then transitory pains appeared in the right leg. After this the pains disappeared from the left leg for about a year, but during the summer of 1909 they again became severe. Indeed, since that time the pain in both legs was constant, though varying in intensity, and on the whole more severe in the right than in the left leg. The pains were described as violent and drawing. It was also noted that when the body was shaken, as when the patient coughed or sneezed, or when she was shaken while riding in a wagon, the pains in the small of the back were made very much worse. The pains were also worse when she was lying down than when she was sitting. They were also increased when she walked or when she bent over. There was no special weakness in her legs, and no paraplegia or paraparesis, and the sphincter of the bladder was in perfect control. Examination showed the left pupil somewhat larger than the right. The pupillary reactions were normal. The pulse was rapid. The back was somewhat stiff, and there was sensitiveness to blows over the lower dorsal and all the lumbar vertebræ. The gluteal muscles were flaccid. The calf muscles were slightly atrophic. Electric reactions were normal. The abdominal reflexes were gone and the tendon-reflexes increased. The right knee-jerks were stronger than the left. The right plantar reflex was present, but the left was very weak, though not absent. There were no sensory disturbances. The *x*-ray showed merely a slight asymmetry of the spine.

A caudal tumor was suspected, and a lumbar puncture done, first between the third and fourth and then between the fourth and fifth lumbar vertebræ. The fluid which appeared came out drop by drop rather slowly under low pressure. It was a clear amber or citron-yellow fluid. In a little while a clot appeared in it. When the clot was removed, another formed. On centrifuging the fluid the lymphocytes were found to be slightly increased. The centrifuged cerebrospinal fluid coagulated completely.

An operation for a caudal tumor was done on the tenth of February, 1910, in the surgical service. At the level of the second lumbar vertebra a tumor about the size of a date which had partially grown to the cauda fibers was found and removed successfully.

Case 24.—The third case was that of a male, thirty-six years of age, who entered the clinic on October 19, 1909. His illness began at the end of July, 1908, with headaches, dizziness, frequent vomiting, and trembling of his arms and body. These changes were merely the beginning changes of a paretic dementia, which can be passed over as being unimportant in this connection. The striking point is that the cord symptoms also began to develop subsequently. His legs became spastic. Pains were complained of in his knees and later in his toes. The pains radiated also to the lumbar spine, and were very violent in character. The blood gave a positive Wassermann reaction. There was also a marked stiffness and pain in the spine and a weakness of the muscles of the abdomen. The patient was unable without support to get up when he was lying down. The upper part of the abdomen was somewhat prominent. The abdominal reflexes were weak, being easily exhausted on either side. The cremasteric reflexes were absent. There was a marked paresis of both legs. The dorsal flexion of the foot was poor. There was both patellar and ankle-clonus, a Babinski, and a suggestion of the Oppenheim reflex, indicating a lesion of the pyramidal tracts.

The lumbar puncture gave issue to a citron-yellow fluid. In a short time it clotted, as it did in the two previous cases. On centrifuging, the clot was found to contain a large number of leukocytes and many lymphocytes. Leukocytes were also found in the overlying fluid. Inasmuch as the case was one of paretic dementia, with subsequent cord symptoms, the cytology of the cerebrospinal fluid was thought to be due to the paretic dementia. The stiffness of the vertebral column, the spasticity of the legs, the weakness of the abdominal muscles, and the double Babinski toe-sign suggested a luetic pachymeningitis hypertrophica. A diagnosis of extramedullary tumor was made, however, on the basis of the two preceding cases. No operation was done, and it is impossible to state whether the diagnosis was correct or not.

Case 25.—Klieneberger's last case was that of a male, twenty-six years of age. Since his nineteenth year he had at times severe pains in his right leg, associated with a dragging of the right foot. With massage and electricity it grew better. At the age of twenty-four and a half he married. In September, 1907, he had a severe case of typhoid fever. After that he always had trouble in emptying his bladder. While convalescent from the typhoid fever he noticed that he was able to move his left hand only clumsily. At the same time he felt a slight pain in it. When he stood on his feet his right leg felt weaker than his left. Moreover, his left arm and hand and right leg were somewhat atrophic. He made a very slow improvement, so that by February, 1908, he was able to return to work.

In September, 1909, he began to have weakness and pain in the neck and a weak feeling in the right toe, followed by cramps in the right leg. Sometimes without any pain whatever it assumed cramp-like movements. When he walked the right leg dragged. In the middle of January, 1910, pains began to appear in his right sole, and the right leg became markedly weak. By the beginning of February he began to have radiating pains in his back. These were not increased by pressure, as in the preceding cases. Later he developed a spasm of his bladder sphincters, so that he had to be catheterized.

Examination on the first of March, 1910, showed a sensitiveness over the sixth to the eighth dorsal vertebræ. Ankle-clonus was present on both sides, more particularly on the left. The Babinski toe-sign was present on both sides. The Oppenheim sign was present on the right side, but absent on the left. Strümpell's anterior tibial sign was present on the right side, but absent on the left, agreeing with the Oppenheim sign. The abdominal reflexes were both absent. The cremasteric reflexes were present. His general musculature was in good condition, though his left shoulder was higher than the right. The spine was somewhat scoliotic, and the lower part was stiff. His left hand showed a slight atrophy of the abductor indicis. Finger adduction and thumb abduction were not executed so well on the left side as on the right. His right leg dragged. Passive motion was not interfered with. There was an ataxia of both legs. The tendon and periosteal reflexes of the arms were increased. There was hyperalgesia in the right and hypalgesia in the left leg. There was also a decrease in sensation over the lower part of the chest, at which point pain sensation was diminished. Muscle and joint-sense in the toes and ankles was markedly affected. There was some nystagmus. Jaw-jerks and arm-jerks were increased.

The diagnosis oscillated between a tumor of the spinal cord and multiple sclerosis. The latter diagnosis seemed the more likely, particularly because of the nystagmus, increased jaw-jerks and arm-jerks, absence of the abdominal reflexes, with well-preserved abdominal muscles, and the presence of the cremasteric reflexes. Moreover, the course of the case itself, the slow progress and insidious onset, its development in a series of attacks with improvement and exacerbations, seemed to support the diagnosis of multiple sclerosis. Moreover, a tumor could not explain the increased jaw-jerks, the nystagmus, the atrophy in the left abductor indicis. The latter, however, might have been due to the antecedent typhoid fever. *X*-ray examination showed that the contour of the eighth to the tenth dorsal vertebræ was not sharp. The discs were not clear. This suggested the possibility of a tuberculous spondylitis.

Lumbar puncture was then done, and under slight pressure a citron-yellow fluid containing many lymphocytes and much albumin was withdrawn. A coagulum immediately formed within it. On this account the diagnosis of an extramedullary neoplasm was made.

An operation was done March 23, 1910, by Hartmann, the laminæ and spinous processes of the fifth to the eighth dorsal vertebræ inclusive being removed. When the dura was opened, much yellow cerebrospinal fluid escaped. At the sixth dorsal segment there were found two bulgings out of the spinal cord about the size of a pea or a bean. When these were excised, a few drops of clear fluid escaped, and then the bulging collapsed on the posterior side of the cord. The pia was found to be slightly inflamed, and it showed little tubercles about the size of a pinhead. When these were examined by Ponfick, he reported that they were made out of fibrin network containing a few spindle and stellate cells, suggesting a specific infection. Hartmann and Klieneberger looked upon the thing as evidently being some sort of cystic disease, which in its symptomatology resembled a tumor.

At the close of his paper Klieneberger apparently makes out, without the aid of the French literature, that in these cases of xanthochromia with massive coagulation the tumor always divides the canal into two parts. The fluid in the lower part is intensely yellow—much more so than after a cerebral hemorrhage. Moreover, the fluid is always perfectly clear and contains no blood. The fibrin coagulation distinguishes it essentially from all other cerebrospinal fluids. Often in a few minutes coagulation takes place, whereas the small coagulum found in tuberculous meningitis often does not appear inside of twenty hours. Klieneberger looked upon the whole affair as probably derived from blood. He believes that the lymph circulation in the spinal canal is cut into two sections either by a tumor or by a meningitic process.

CASE 26.—Another case reported in German literature and mentioned by Klieneberger also appears in Mestrezat's book on the cerebrospinal fluid as a *cas fruste* of this syndrome. After reading the report I am rather inclined to look upon it as belonging to the syndrome without qualification. Walter Rindfleisch's publication appeared in the *Deutsche Zeitschrift für Nervenheilkunde*, 1904, vol. xxvi. Klieneberger is not inclined to look upon Rindfleisch's case as belonging to this syndrome, because the yellow color was not so intense, the albumin was not so abundant, and the tendency to coagulation was less marked. The fluid which was obtained in Rindfleisch's case, which was one of sarcomatosis of the spinal cord, was a dark yellow or brown, and contained a number of cells, but no red blood-cells. The clot did not form until some hours, and the amount of albumin present was 2.4 per cent.

In addition to these 26 cases 7 more have been reported, 4 by Marinesco and Radovici, 1 of which came to postmortem; 1 by Debres and Paraf, and 1 by Launys, Froin, and Ledoux. This last case was reported in the *Gazette des hôpitaux*, February 24, 1914, and to the best of my knowledge and belief is the last case recorded as such. A seventh case has been reported in American medical literature by Joseph Collins, of New York, making 33 in the entire medical literature up to the present time.

The 4 cases reported by Marinesco and Radovici were recorded in the *Revue neurologique* for February 6, 1913, p. 268. These authors called attention to Froin's cases in 1903, and to Mestrezat's collection of 21 cases. They then proceeded to report 4 cases of their own, differing in the etiology and clinical history, but all agreeing in the syndrome of xanthochromia, massive coagulation, and hematolymphocytosis. The cases were reported imperfectly.

Case 27.—The first, a male, forty-five years of age, a drinking man, denying venereal infection, entered the hospital March 14, 1912, with paraplegia and contractures, the onset having been seventeen years before. The patient could neither walk nor stand. The tendon-reflexes were all exaggerated. The cremasteric and abdominal reflexes were absent. The Babinski toe-sign and ankle-clonus were present. Tactile, pain, and thermal sense were lost below a line drawn 2 cm. below the navel. The Wassermann reaction was negative on both the blood and cerebrospinal fluid. Six punctures in all were made during a period of six weeks. In all of them xanthochromia was found, and in some of them the coagulation tendency was so great that coagulation took place in the needle. In one specimen obtained five weeks after the first the fluid was opalescent and did not coagulate. The period of coagulation in all the specimens varied from immediately to as high as two hours, and even two and one-half hours. The cerebrospinal fluid removed at the level of the seventh and eighth dorsal spines showed no xanthochromia, coagulation, or lymphocytosis. The patient at the time of the reporting of the article was in the same condition as at the time of his entrance into the hospital.

Case 28.—The second patient, a male of thirty-six, also showed a complete paraplegia with increased tendon reflexes and ankle-clonus, besides the Babinski toe-sign in the right leg. The cremasteric and abdominal reflexes were present. Tactile, thermic, and algesic sensations were gravely affected as high as the twelfth rib. There were, furthermore, incontinence of urine and obstinate constipation. Lumbar puncture showed a citron-yellow fluid under low pressure, coagulating in two hours into a jelly-like mass. Death took place January 15 from "surgical kidney." Postmortem examination from the second to the eleventh dorsal segments showed that the cord and meninges were increased in volume, that the meninges had grown completely to the cord. So complete was the union that one could not detach the meninges without injuring the cord. The terminal portion of the cord and the beginning of the cauda were completely enveloped in a mass of inflammatory neoformation.

Case 29.—The third case, a male of thirty-two, entered the hospital June 28, 1911, having been ill four months. His sickness began with pain in the lumbar zone and, deeply situated, in the left thigh; also weakness of the limbs. In a month the progress of the disease had been so rapid that he was forced to his bed. Over the left lumbar region, particularly over the last dorsal and first lumbar vertebræ, appeared a swelling which was painful to pressure. The paraplegia was flaccid. The patellar, Achilles, and cremasteric reflexes were absent. There was no Babinski toe-sign. There was an extensive atrophy of the thighs and calves. Sensation was absent as high as the two false ribs. There was a retention both of urine and of feces. Lumbar puncture gave rise to a citron-yellow fluid, coagulating within a few minutes and containing a few lymphocytes. Puncture of the tumor mass showed only blood. This tumor mass proved to be an osteosarcoma of the vertebral column. A month later a trophic condition developed, the man being taken home almost moribund, postmortem examination being refused.

Case 30.—The fourth case was a male, thirty-eight years of age, who went into the hospital December 24, 1911. This patient was given an intravenous injection of stovain November 20, 1911, and a phimosis was operated upon. Three days later he began to have trouble in walking, and in a few days was obliged to take to his bed. By December 15 both of his arms were involved. At this time he showed almost a complete flaccid paraplegia, and the movements in his fingers and other joints in the upper extremities were reduced. His face was expressionless, and there was a mild paresis of the right facial nerve. His knee-jerks, Achilles jerks, his triceps, biceps, and trapezius responses, were absent. Cutaneous reflexes were also absent. Sensory disturbances were very slight with the exception of severe pains in his legs, these pains being increased by pressure on the nerve-trunks. Sphincter disturbances also appeared. A lumbar puncture gave rise to a fluid, which was greenish-yellow, but coagulated within two and one-half hours. By the end of January, 1912, the patient began to get better and subsequently he entirely recovered.

These four cases show the conditions which thus far have been assigned to this syndrome. Two are cases of meningomyelitis, one was a sarcoma, and one was a true meningeal affair, under the picture of a Landry's paralysis. The only one which came to postmortem showed a dural pocket, attention to which was first directed by Sicard and Descomps. Thus, as Marinesco and Radovici showed, chance permitted them to gather together in four cases, three pathologic conditions which lead to massive coagulation and xanthochromia. The third case leads to the question of the effect of a neoplasm upon the cerebrospinal fluid. Marinesco and Radovici did not attempt to explain the pathogenesis of this condition, but they believed it due to some kind of specific degeneration of the vascular epithelium.

CASE 31.—A case reported by Robert Debré and Jean Paraf appeared in the *Presse médicale*, November 26, 1913. Their patient, a girl fourteen years of age, showed an acute meningitis. It is not necessary to review the symptomatology, which was that of a typical tuberculous meningitis. A lumbar puncture was done immediately upon her entrance to the hospital, September 9, 1912, and 10 to 15 c.c. of a citron-yellow fluid, very viscous, ran out drop by drop. A clot began to form within fifteen minutes' time and to separate within an hour. Abundant cells, 383 per c.mm., consisting both of red blood-cells and leukocytes, began to separate out in the serum. A differential count of the white cells showed 22 per cent. of polymorphonuclear leukocytes, 26 per cent. of medium and large mononuclears, and 52 per cent. of lymphocytes. There were no bacteria present.

On the following day a lumbar puncture was again done. The white cells showed 63 per cent. of polymorphonuclear leukocytes, 3 per cent. of large and small mononuclears, 24 per cent. of lymphocytes. There were numerous red blood-cells. Coagulation was immediate when a drop of human serum or a small quantity of calcium chlorid was added to the fluid. Left to itself, coagulation took place spontaneously within ten minutes. On September 16 a lumbar puncture was again performed, and a guinea-pig was inoculated. This pig died January 14, 1913, showing tuberculosis of the liver and spleen. This case is interesting because it is apparently a case in which tuberculous meningitis was the cause of the xanthochromia, coagulation *en masse*, and lymphocytosis. These authors agree with the others who have written upon the subject in believing that the syndrome is due to the closing off of the lower cavity in the spinal canal. This, they think, was brought about by meningeal adhesions.

Case 32.—The last case in French literature, already referred to, is that of Launys, Froin, and Ledoux, entitled, "*Un cas de syndrome de coagulation massive du liquide céphalo-rachidien avec autopsie. Blocage du cone dural par symphyse méningo-médullaire de nature syphilitique.*" These authors called attention to the 21 cases reported by Mestrezat, to which were added the 4 by Marinesco and Radovici and the 1 just mentioned by Debré and Paraf. To these 26 cases the authors add another, making 27. Their review of these 27 cases is very thorough, and it is evident, from their case, that the syndrome is found in the course of meningitis, meningeal tumors, and especially in the cases of meningoradiculomyelitis with paraplegia. Their case was one of meningomyelitis syphilitica with an obliterating meningeal concrescence and a blocking off of the dural cone. It occurred in a laundress, forty-eight years of age, who entered the hospital November 25, 1912, because she could not walk. There was no hereditary taint except that a brother had died of phthisis at the age of twenty. Four months before the patient entered the hospital she had a sharp pain in the right ankle one morning on arising. This pain spread in the next few days to the calf and knee. Next the limb became numb and weak on walking. Two months later the left thigh became involved, and she was able to walk only when holding on to the furniture. One month previous to entering the hospital the lumbar spine was very painful and the right leg completely powerless. For a week the pain had been so great that it finally brought her to the hospital.

Examination showed the right leg completely paralyzed, with the exception of the adductors, which contracted slightly, though not sufficiently to move the leg. Passive motion was possible and painless. The knee-jerks were gone on both sides, and neither the Babinski toe-sign nor ankle-clonus was present on either side. Sensation to the pin-point was diminished on the right side, especially in the perineal branch of the sciatic. The thermal sense was normal. There was retention of feces and also incontinence of urine. The reflexes in the upper extremity and the pupillary reflexes were normal. Physical examination showed the sacrum to be painful in the midline, as though there were a sarcoma involving the sacro-iliac joint, because on pressing together the iliac crests the pain was lessened.

The progress of the case was rather gradual. The left leg also became paralyzed, so that the monoplegia gave place to a complete flaccid paraplegia. Subsequently trophic disturbances appeared, with a slight edema of the lower extremities, especially in the right and median sacral trunk. The incontinence of the urine became constant. The hyperesthesia gave place to a right hypesthesia. Finally a slight Babinski sign developed in the left leg. During the month of December the patient gradually grew worse and death took place January 25, 1913.

Several lumbar punctures were performed. On November 27 the fluid flowed drop by drop. It was yellowish-green in color, and coagulated in one to two minutes. No microscopic examination was possible on account of the coagulation. Cultures were made and no growth took place. On December 27 a lumbar puncture was again performed, and the fluid gathered in a sodium citrate solution. This did not coagulate, but the fluid collected in another tube coagulated at once. Lymphocytes were found in the fluid in the sodium citrate solution. January 11 the cerebrospinal fluid was limpid and flowed readily. Four tubes were collected, the first of which was limpid, the second yellowish, the third rather yellow, and the fourth limpid. None of them coagulated, but they contained lymphocytes and red blood-cells. On January 19 the fluid was limpid, but showed additional lymphocytosis. The Wassermann made was negative. Thus, of the four punctures, two showed coagulation and two did not.

The postmortem examination showed a normal liver, heart, kidneys, and spleen, though the liver was a little large and somewhat yellowish. The spinal cord was normal except at the lumbar area and the conus terminalis, where was found a swelling, regularly ovoid, the size of a pigeon's egg, 5 to 6 cm. long and 3 to 4 cm. broad, soft, and semi-fluctuating. The dura was intimately fastened to the right surface of the swollen area, and very slightly to the left. The involvement of the subarachnoid space was caused by a fibrous concrescence of the dura mater and the cord on the right side, and on the left by an inflammatory adhesion of the meningeal surface. The adhesion on the left was easily broken by slight traction. The dural cone was changed into a subarachnoid pocket, a lymph-tube, by the concrescence. The outer surface was smooth and apparently normal. Within, the cauda equina seemed normal except at one point, where a little tissue formation involved a few roots on the right side, about 3 cm. long and 1 cm. wide. This area was hard and resistant to pressure, which was caused merely by the fusion of the dura mater and roots. A cross-section showed an inflammatory process gluing together the dura and conus terminalis in the center.

Microscopic examination showed that the tissue which agglutinated the roots and the dura was meningeal. Furthermore, there was a diffuse radiculomeningitis with abundant lymphocytes. The arterioles showed a chronic endarteritis and also a periarteritis. The caudal roots were agglutinated with tissue infiltrated with lymphocytes. The disease had spread also into the anterior and posterior median fissures of the cord. The cord itself was edematous. There was a chromatolysis of some of the cells. There was also a diffuse proliferation of the neuroglia and an obstructed ependymal canal. In general the microscopic diagnosis was a gummatous meningomyelitis. The little tumor involving the roots of the cord above mentioned consisted of connective tissue with lymphatic and plasma cells, evidently syphilitic. The authors, therefore, concluded their case to be a meningeal gumma with a meningomyelitis and a meningoradiculitis syphilitica.

This case is classic as to symptoms and findings. It shows a progressive paraplegia with death, as did most of the cases reported. This is the eighth case in which an autopsy has been made.

These authors believed that the yellow color of the cerebrospinal fluid is due to a local hemolysis or to an exudation of the blood plasma. It is not due to a hemorrhage, for the cerebrospinal fluid in a hemorrhage into the ventricles of the brain is fluid in appearance, has no fibrin, and does not clot. Babinski, it will be remembered, explained the color as due to a fibrinous hemorrhagic meningitis, but it has been learned that in hemorrhagic meningitis the fluid is red, with very little fibrinous coagulum, and never shows coagulation *en masse*. In all the 27 cases known to these authors in the literature, the color of the cerebrospinal fluid is more or less yellow or greenish yellow, never red or rose, such as characterizes hemorrhagic fluid. Some other explanation than hemorrhage must be advanced. Apparently the blocking off of the dural space is very important.

The eight autopsies which have been made on these cases, some of which have already been mentioned, may very briefly be reviewed:

1. Cestan and Ravaut (1904). At the level of the conus terminalis there was found a fibrous concrescence of the meninges and cord caused by an internal annular pachymeningitis.

2. Tedeschi (1906). There was a swelling at the level of the conus terminalis and slightly above it, with a rather extensive meningomedullary concrescence, enveloping the corresponding part of the conus terminalis and of the cord, as well as all the roots.

3. The case of Sicard and Descomps (1908). The thickened dura mater formed a true perimedullary sheath from the tenth dorsal to the second sacral segments of the cord. In places an agglutinating process had fastened together the dura and pia mater and enveloped the cord in a hard connective-tissue sheath.

4. The case of Blanchêtière and Lejonne (1909). At the level of the eighth and ninth dorsal segments a meningeal sarcomatous tumor was found which probably acted as a ball-valve and cornered off the subarachnoid space, causing a dural pocket below.

5. The case of Verhoogen and Dustin (1910). This case showed a compression of the cord from Pott's disease, and a sleeve

of pus surrounding and compressing the cord over an extent of several centimeters.

6. The case of Mestrezat and Roger (1912). At the level of the eighth and ninth dorsal vertebræ a pachymeningitis compressing the cord over quite a considerable area. Above and below this level the cord was edematous and strangulated by a very tight meningeal sheath, cutting off all communication between the lumbar culdesac and the space above.

7. The case of Marinesco and Radovici. Between the second and eleventh dorsal segments the cord and the meninges, which latter were increased in volume, were massed together, so that the meninges could not be separated from the cord. The conus terminalis and also the cauda were enveloped in an inflammatory mass of neoformation.

8. The last is the case of Launys, Froin, and Ledoux. This case is one of the meningomedullary concrescence on the right and an inflammatory adhesion of the meninges on the left of the cord near its lower end.

All the autopsies reported show that there is an interruption, more or less complete, in the subarachnoid space, with a complete or partial blocking-off of the conus terminalis or a less complete blocking-off of the dural cone. One can easily understand why the cerebrospinal fluid cannot get down into the culdesac in the lumbar area, and why, therefore, the contents of this culdesac differ from the cerebrospinal fluid above. This difference is not theoretic, but real, because Marinesco, in one of his cases, obtained a yellow fluid from the dural cone and a limpid normal cerebrospinal fluid in the upper dorsal meningeal canal.

Probably the pouch alone is not enough to give rise to the syndrome. The pocket may, possibly, by reason of the diminished pressure within it, have been filled with an exudate which is almost pure blood-serum. That it is an exudate of this type seems rather to be suggested by the very presence of the red blood-cells, together with polymorphonuclear cells and large and small lymphocytes. We personally do not believe that the hemorrhage has any part in the formation of this peculiar cerebrospinal fluid. However, the characteristic pouch is a *sine qua non*, and probably into this pouch for some reason or other the exudate of blood-serum takes place. It is not necessary that there be an inflammatory process, because the exudate has occurred in tumor cases.

Case 33.—The only case reported in American literature appeared in the *American Journal of the Medical Sciences* for April, 1914, in a report by Joseph Collins and Charles A. Elsberg on three cases of combined tumors of the conus and cauda equina. Their second case belonged to this syndrome, although not specifically mentioned as such. The patient, a woman, a Russian Hebrew, aged twenty-five years, married, complained of pains in the back radiating down the legs, more severe in the left than in the right leg; weakness of the lower extremities and difficulty in passing urine. The duration of the symptoms had been about two and one-half years, the initial symptom being pain. This pain began about a fortnight after she married, gradually increased in severity, and since the birth of her child, ten months after marriage, it had grown rapidly worse. The pain was increased by any attempt at movement, such as walking or going up and down stairs, and was less when lying down. She had been subjected to many kinds of treatment; she had worn arches for double flat-foot, and had been in a plaster jacket for her spinal trouble.

Physical examination showed her station weak and uncertain in strength. There was a drop-foot on both sides. The lumbar spine was curved. The knee-jerks were feebly elicited. The ankle-jerks were present. There was no Babinski phenomenon. Abdominal and epigastric reflexes were present. Sensation was impaired along the inner and posterior aspects of the right thigh and over the whole of the right leg and foot. There was also a drawing sensation over most of the left leg and all of the left foot. There were disturbances of touch, temperature, and pain. "Laboratory examinations were all negative," save for the lumbar puncture, which gave a dark-yellow fluid which contained no cells and coagulated solidly on the application of heat.

This case very likely would have shown spontaneous coagulation of the cerebrospinal fluid if it had been tested from that point of view. I feel sure, on account of the solid coagulation by heating and on account of the fact that the case was one of tumor of the cauda equina, that the case belongs in this syndrome. A laminectomy was done December 3, 1910, and the spinous processes and laminæ of the third, fourth, and fifth lumbar vertebræ and all of the second and first lumbar and twelfth and eleventh dorsal vertebræ were removed. Collins notes that the dura was of fleshy color and that the cord did not pulsate. The dura was incised, exposing a large, reddish-brown tumor mass which surrounded the roots of the cauda equina and filled up the entire spinal canal to the lowermost part. It was on this account that it was necessary to remove the eleventh and twelfth dorsal and the first and second lumbar vertebræ, and to excise the dura upward before the upper end of the tumor was exposed. The growth extended upon the conus and lumbosacral cord, and was but slightly adherent to the cord. During the various manipulations the tumor gradually bulged out of the canal until it seemed about three times as thick as when first exposed. Below could be seen some roots of the cauda equina surrounded by caudal tissue. It seemed advisable to delay the removal of the growth in order to allow the tumor to extrude from the canal. The wound was therefore closed without drainage.

A week later the wound was opened and the tumor was found markedly extruded. It was removed from the conus and from the inner surface of the dura. It was very large, smooth, and glistening. The growth was easily freed from the roots of the cauda, though it was adherent to the conus terminalis. The patient made an uneventful recovery, so that by September 6 of the following year she could walk, though she had trouble on account of the persistent flat-foot. She was free from pain, and no disturbances of sensation could be found, though the knee- and ankle-jerks were still absent.

Undoubtedly in this case the tumor exercised a ball-valve action, there being no inflammatory adhesion on the inner surface of the dura, which is described in the report as very large, smooth, and glistening, but the fluid showed xanthochromia and massive coagulation on heating.

CASE 34.—The present case, which I believe to be the thirty-fourth to be reported, entered Mercy Hospital November 6, 1914, in Dr. Murphy's service.

History.—The patient is a man, sixty-eight years of age. He has had the usual diseases of childhood. At the age of fifteen years he had typhoid fever and was sick four weeks. There were no complications. His family history is negative as to both tuberculosis and carcinoma. He has eight children living and well. He denies ever having had a venereal infection of any sort.

Thirty-five years ago the patient was pinched between a wagon and a log, which caught him in the small of the back and caused severe pain at the time. He was laid up two or three days with a lame back, but does not remember much about the condition. Thereafter he has always had lumbago, coming on at varying intervals.

Fifteen years ago the patient had a severe pain in the right hip, back of the right thigh and calf of the right leg. The pain was sharp and shooting in character and paroxysmal in type. The onset was very sudden after he had helped to carry in a load of furniture. The next day he was confined to bed and remained there three months. He had no chill or fever accompanying the attack. After three months in bed he got up greatly relieved, suffering no further trouble other than an occasional attack of lumbago for five years.

At the end of this time he noticed a sharp pain at the right lower costal margin, radiating to his right shoulder, associated with nausea and vomiting. At this time he noticed an icteric hue in the whites of his eyes. The attack lasted one hour. Ten days later he had an attack with a similar train of symptoms. There was no chill or fever to his knowledge at either time, but he had profuse night-sweats. His attending physician called the condition "gall-bladder disease."

During the following nine years he had an occasional pain between the shoulders and in the hips, but it never incapacitated him.

In August, 1912, the patient had severe attacks of pain in the right hip, the back of the right thigh, and the calf of the right leg, as above described. The motion in the hip was good, but was very painful during the paroxysms. This continued at varying intervals up until two months ago, when he noticed severe pains in the lumbar and sacral region of the back. Subsequently the above-described pains seemed to attack the left hip, the back of the left thigh, and the calf of the left leg. This condition became persistent, and the spine became very tender to pressure. He was unable to lie on his back or right side on account of the pain.

For the past ten days the patient has taken as much as five grains of morphin sulphate hypodermically in twenty-four hours, yet this amount would not give him much relief, so intolerable was the pain.

The patient has lost 35 pounds in the past ten years. Twenty pounds of this has been lost since August, 1912.

Examination of this patient showed very few physical signs. He came complaining of a sciatica which was present at various times over a period of many years. Interesting in the history is the story of an injury thirty-five years ago in which the patient was pinched between a wagon and a log, which caught him in the small of the back. We are unable to associate that accident with the patient's present trouble. The first sign of disturbance in this patient's case occurred fifteen years ago, when he had a severe pain in the right hip On that occasion a diagnosis of sciatica was made, and from that time to the present he has had recurrent attacks of sciatica. His real acute disturbance did not, however, begin until August, 1912, at which time he began to have pain, which was of the typical root-pain character, and distinctly neuralgic, often sharp and stabbing in character, but occasionally settling down to a kind of dull ache. So intolerable did these pains become that the patient finally began making use of morphin to control them, and so ineffectual was the morphin that at the time of entrance into the hospital the physician who treated him told us he was giving the patient between four and five grains without very much relief of the pain. Before entering Mercy Hospital the patient furthermore had three injections of water into the right sciatic nerve, this having been done for the purpose of relieving what was supposed to be his intense sciatica.

When he entered the hospital not only was he complaining of pain in his right leg, but he was also beginning to have pain in his left leg. Moreover, he was beginning to manifest a certain amount of weakness, so that he was using crutches on his entrance.

His knee-jerks were present, but very much reduced. It was not necessary to employ reinforcement to get them, but it was necessary to have the leg in a very favorable position in order to get the responses with the hammer. His ankle-jerks were also present but diminished. His cremasteric and abdominal reflexes were present and apparently unchanged. There were no subjective or objective sensory findings, either as to touch, temperature, or pain. Muscle and joint sense and sensibility were not involved in any way. The pain was very intense in the sciatic territory of the right limb, for the most part in the right leg, but on careful examination occurring also in the anterior crural territory, and also in the territory of the ilio-inguinal and iliohypogastric nerves. The pain in the left leg was confined generally to the sciatic region of the thigh. There was a distinct atrophy of the quadriceps extensor on the right side, and there was a distinct paresis of both legs. The case, therefore, presented itself at a time when the symptoms were relatively paraparetic as to the motor roots, and irritative rather than paralytic as to the sensory nerves. The sensory nerve-roots involved were those of the ilioinguinal and iliohypogastric nerves, the anterior crural nerve on the right side, and both right and left sciatic nerves. This indicated a lesion extending as high as the first lumbar roots. He did not give a trace of disturbance of the reflexes of the upper extremities. The pupillary reflexes and the cranial nerves were all perfectly normal. The man himself did not look cachectic, though he was plainly suffering. To be sure, he had lost a good deal of weight by reason of loss of sleep and loss of appetite, rather than by reason of any metabolic, infectious, or neoplastic condition.

In the presence of a double sciatica apparently due to root-pains a luetin examination was promptly made, with absolutely negative findings. Recourse was then and there had to a lumbar puncture, and to our great surprise we obtained a yellow fluid. It came out rather promptly under average pressure, and was collected in a test-tube. Within a half-hour the fluid had coagulated so that the test-tube could be turned upside down and not a drop of the fluid escaped from it. It was a perfectly gelatinous mass. It was put in the ice-chest, and by the following morning the clot had partially separated. A Wassermann test was made on the cerebrospinal fluid on the following day and found weakly positive. Because of the fact that the patient gave an absolutely negative luetic history, was the father of eight healthy children, that there had been no miscarriages in the family, that there never had been a symptom in any way suggestive of a luetic infection in his case, the positive Wassermann reaction was regarded as due to some other cause than a luetic one. The serum which escaped from the contracting coagulum was centrifuged and found to contain about 40 cells per field. These were mostly lymphocytes. The clot itself was also examined and found to contain some cellular elements.

Following the lumbar puncture a remarkable thing happened. The next morning the patient was reported well. He had slept very well the night before, and the pain had entirely disappeared and he was able to sit up in a chair and take a step or two. Within two days the pains reappeared, and again a lumbar puncture was done, but this time we had great difficulty in getting the fluid. The needle was inserted seven times before the fluid could be obtained. At the time we could not account for this condition, but subsequently it became rather evident. Examination of this fluid, which coagulated very rapidly, disclosed the same cellular elements as the first day. The Wassermann reaction this time was negative. Lange's colloid reaction was also negative, and the Noguchi reaction was negative.

Again the relief of pain followed, this time lasting only twenty-four hours. This relief of the pain suggested the possibility that we were dealing with a cyst. It brought to mind the possibility of a meningitis serosa circumscripta of the conus. Recalling, then, the original article of Cestan and Ravaut which I [Mix] had read in 1904, I began a study of the medical literature, which I have reviewed.

Having arrived at this point in the study of the case, it was evident that xanthochromia with coagulation *en masse* and the presence of hematolymphocytosis occurs in three types of cases. The first type consists of certain acute cases, probably all of them meningitic, two of them in the literature existing under the guise of Landry's paralysis. In these cases it would seem that there is probably an agglutination somewhere in the lower dorsal or upper lumbar part of the spinal canal, where the pial and dural pocket is formed, so that proper conditions for xanthochromia, coagulation *en masse*, and lymphocytosis are present. As the meningitis subsides and the transitory agglutinations dissolve, the cerebrospinal fluid changes back to its normal type. These cases may or may not recover. If, however, as in the case reported by Debré and Paraf, the meningitis is a tuberculous meningitis, the case will, of course, go on to death, though the cerebrospinal fluid may vary from time to time in color and coagulation.

The second type of case is that of a paraplegia of either the flaccid or spastic type, depending upon whether the process be confined to the cauda or to the lower part of the cord and the cauda. In most of these cases there has been a pachymeningomyelitis of the conus terminalis. It has always been an internal pachymeningitis, the outer surface of the dura being in every case intact. In this type of case there is an agglutination of the roots and of the dura, and the arachno-pia. Generally the subarachnoid space of the dural cone is entirely cut off from the space above. However, in these cases it is evident that the cutting off by adhesions, agglutinations, and concrescences is oftentimes so imperfect that one may find not infrequently an alternation between normal cerebrospinal fluid and fluid showing the syndrome of xanthochromia, massive coagulation, and lymphocytosis.

The symptomatology in these cases is polymorphous, but the localization is always in the spinal canal at the lumbar level. It may involve only the cauda, or it may involve the conus terminalis or the lower part of the spinal cord. Consequently the cases almost always show a paraplegia, but the paraplegia may be spastic or flaccid. Sphincteric disturbances are very apt to be present, though not necessarily so. Sensation is almost invariably disturbed. Pain is very severe.

The third group of cases comprises the tumors. This group is undoubtedly made up of two sorts of tumors. The tumors of the first variety have neoplastic formations about them, especially granulomata and sarcomata, limiting themselves to the dura and pia, and dividing the spinal canal into two compartments—an upper, containing clear fluid, and a lower one, containing yellow, coagulable fluid. The second sort of cases is made up of tumors which apparently cut off the lower portion of the spinal canal by a *ball-valve* action, without adhesions. This kind of a cutting-off of the lumbar culdesac has never been mentioned in the literature. Undoubtedly the case reported by Klieneberger and our own case of tumor of the cauda equina of a cystic, smooth variety belong in this category. In Blanchêtière and Lejonne's case, though the authors themselves did not call attention to the fact, the osteosarcoma found at the autopsy probably acted as a ball-valve, completely cutting off the subarachnoid space above from that below This is the explanation why, in this particular case, the cerebrospinal fluid in all the punctures was always yellow, and always showed the characteristic signs of the syndrome. In this respect it agreed with our own case, which also showed in the four lumbar punctures made precisely the same findings as to color, coagulation, and lymphocytosis. This case, moreover, agrees with our own in another respect, the comparatively small number of lymphocytes. This case showed but two or three lymphocytes to the field and ours about 40 cells to the field, whereas in an inflammatory case 300 or 400 cells to the field have been usually found.

In our case we did not make a positive diagnosis of tumor. We, of course, knew that the case with which we were dealing did not belong to the first group of acute cases. But did it fall in the second group, in which there was an inflammatory pachymeningomyelitis internus of the conus terminalis, or in the third group, in which a tumor might act in much the same manner as the osteosarcoma acted in Blanchêtière's case? Operation was, therefore, determined upon, and, so far as I know, this is the fourth case of this syndrome which has ever been operated upon, the other three being two of Klieneberger's and one of Collins.

Blanchêtière and Lejonne regretted that an operation had not been done in their case, closing their article by stating: "It is probable that in our case an early operation would have saved the patient."

In the present case, believing that either a tumor or pachymeningitis internus was present, the question of operation came keenly to the front. It was felt that possibly an operation might be a benefit, and in the event that a pachymeningitis internus was present, that the operation devised by Dr. Murphy might be performed, as will be stated in his portion of this article; and in the event that a tumor was responsible for the disturbance, that the tumor might be removed. The patient was put into complete possession of the facts, and the medical literature was gone over with him. He very readily consented to an operation, the results of which are mentioned in Dr. Murphy's discussion.

Physical and Chemical Characteristics of the Cerebrospinal Fluid.—Analysis of the cerebrospinal fluid in all the cases with this syndrome shows two absolute essentials: First, is the spontaneous or provoked perfect massive coagulation due to the presence of an abnormal constituent, namely, fibrin, in the fluid. The consistence of the clot may be variable, but often it is so solid that one can turn the test-tube upside down without a drop being spilt; in other words, it completely gelatinizes. In some instances the case may show a complete coagulation of the cerebrospinal fluid at one puncture, and a partial coagulation with a large mass of coagulum floating in the fluid at another puncture.

The second absolute necessity is the yellow color. Generally this yellow color is very pronounced, but does not have within it a shade of brown. This brown shade, shown by the fluid, which I myself have observed recently in a case, is usually due to hemoglobin. Rose at first, it later turns into a brownish-yellow.

The third characteristic which this fluid shows, as originally described by the French, is hematolymphocytosis. This characteristic is not a necessary part of the syndrome. We find, for example, two groups of cases, the first belonging to cases of meningitis, Landry's, meningomyelitis, and some of the granulomatous and sarcomatous tumor cases, showing an increase of the cellular elements, and other smooth tumor cases showing a perfectly normal cellular content. *In general, if the fluid shows a normal cytology, it is likely that it belongs to the ball-valve tumor type.* If the fluid, on the other hand, shows lymphocytes, then it probably belongs to the meningomyelitic, meningeal, or inflammatory neoplasm type. I am strongly of the opinion that the presence or absence of the cellular constituents is a very positive differential diagnostic point.

The tension of the fluid is remarkably variable. In some of the cases it is high. In others it is so low that only a few drops run out. The pressure varies at times in the same case. That was true of our own case. On the first occasion the fluid was under considerably more pressure than it was on the second occasion. On the third occasion the pressure had again increased.

The appearance of the cerebrospinal fluid is always the same. It is perfectly *limpid*. Notwithstanding the fact that it contains a large amount of albumin, relatively it contains a small number of cells. The consistence is usually thin and watery, but sometimes it is rather thick, as in Donath's case, and also the case reported by Fornaca. The *color* is variable both in intensity and in shade, but it is always plainly yellow. It has been reported as greenish-yellow, as brownish-yellow by Sicard and Descomps, and in Donath's case as having a reddish tint in addition to the yellow. One possibly can note in these various tints a succession of shades which are observed in ordinary hemoglobin decomposition products. Pigment found in the fluid has been identified as hemolytic biliary pigment by Donath, biliary pigment by Blanchêtière and Lejonne, and urobilin by Fornaca.

The specific gravity is high,—usually 1019 or 1020,—but a specific gravity as low as 1012 has been reported by Blanchêtière and Lejonne.

Albumin is very large in amount. In no other condition is the cerebrospinal fluid found to contain so much albumin as in this syndrome. Tedeschi reported 2.88 per cent.; Fornaca, 4.17 per cent.; Blanchêtière and Lejonne, 2.55 per cent. to 2.70 per cent., and Mestrezat has found even 4 per cent. present.

Fibrinogen and fibrin are its most characteristic constituents, probably the spontaneous coagulation being the most typical and striking thing about it. Sometimes coagulation takes place in the needle itself, as in the cases reported by Froin, Sicard and Descomps, and Mestrezat. Usually it appears in ten to twelve minutes, sometimes in half an hour, and sometimes after hours. At times there may be no spontaneous coagulation even after a long period of time, but coagulation can often be induced by the addition of two drops of blood-serum. One may be able to turn the tube upside down without spilling its contents, or it may contain at times a comparatively small coagulum. Usually on standing the clot separates out from the fluid just as a blood-clot separates out from blood. The fibrin has been estimated by Fornaca as 0.6 grain per liter; by Blanchêtière and Lejonne as 1.7, 1.66, 1.63 grains per liter. Derrien, Mestrezat, and Roger found in one of their cases 0.65 grain per liter. These values well approach that of the fibrin in the blood, and are pathognomonic of the syndrome. The other constituents found in the fluid are albumoses, sugar, urea, and small amounts of chlorids.

Pathogenesis.—The pathogenesis of these cases has not been very accurately developed. Babinski associated the idea of hemorrhage with this syndrome as early as 1903; and Cestan and Ravaut, in 1904, were disposed to agree with him. In 1908 Sicard and Descomps regarded the fluid as rather a transudate. These authors, though they did not grasp the idea of a closed-off culdesac at the lower end of the spinal canal, nevertheless insisted on the presence of the meningeal pockets of an agglutinative origin, and to this they added the idea of a transudate. Derrien, Mestrezat, and Roger claim the honor of first enunciating the culdesac hypothesis. An analysis made by them of their case and all cases reported up to 1909 led them to their conclusion. They modestly confess, however, that the idea of a closed-off cavity could not have been entertained earlier, for it could occur to observers only after numerous cases had been analyzed and compared with one another and the chemistry of the fluids studied. This was done first in 1909. The syndrome depends essentially upon two conditions—first, on the existence of a closed cavity isolated from the general cerebrospinal cavity in which elements from the blood may accumulate, either from hemorrhage or a transudate. Second, an alteration (infectious, toxic, or neoplastic) of the blood-vessels of that culdesac, creating a transudation, or leading to multiple microscopic hemorrhages, as is doubtless the case in patches of pachymeningitis, vascular tumors, etc.

Diagnosis.—Whenever the cerebrospinal fluid is found to conform to this syndrome, it means that there is an isolated lumbar culdesac. It may be isolated by a meningitis, which has sealed the meninges to the surface of the cord, or it may be due to a pachymeningomyelitis, or to a tumor which compresses intradurally or extradurally, and so cuts off by pressure the culdesac, or to an intradural tumor which acts as a ball-valve, plugging up the top of the lumbar enlargement of the spinal cavity. It always means an organic disease: it never means hysteria.

As to the therapeutic value, Mestrezat in his book shows that:

First: one must regularly evacuate this cavity by systematic lumbar punctures. He has succeeded very well in his cases by this process.

Second: Nothing should ever be injected therapeutically into the subarachnoid space in such a case because, in his experience, reabsorption does not take place from such a meningeal pocket.

I would rather say that there is no therapeutic value in the removal of such fluid. It at once returns, as in our own case. Cases of Landry's type and the non-operative cases must be treated medicinally, of course, and if of the leptomeningitic type, provided it be not a tuberculous leptomeningitis, with considerable hope of ultimate success. The meningomyelitis cases and the tumor must be treated operatively. The meningomyelitis cases may be treated by an operation, which Dr. Murphy in this article for the first time proposes. The tumor cases, which can oftentimes be diagnosticated by the development and course of the case, by the occurrence or absence of cells from the cerebrospinal fluid, should always, of course, be operated upon. It has been shown by several cases that many of these tumors can be easily removed. Our own was removed with extreme ease, and led to a very gratifying result.

Looking upon these cases as a whole, it would seem to me that, in the presence of xanthochromia, coagulation *en masse*, and lymphocytosis, provided the case is not an acute one belonging to the Landry group or to the acute meningitic group, the case ought to be operated upon; provided further that it is not a syphilitic pachymeningomyelitis. If a luetic infection and tuberculosis can be positively excluded, then the case, without waiting for grave symptoms to develop, ought to be operated upon at once. There is always the possibility of a tumor being found in such a case, as in the case just reported, and there is further the possibility of affording relief provided the case be not one of tumor, but, instead, of pachymeningomyelitis internus of the conus, and non-syphilitic.

Imperfect or masked cases (*les cas frustes*)—Mestrezat reports nine of these cases under three headings.

First: Those showing the absence of xanthochromia, in which belongs Lépine's case, reported in 1903, and the case of Froin's, communicated orally to Aubrey and recorded in his *Thèse de Paris*, 1909.

Second: Cases in which the cytologic formula occasionally fails, even in the most typical case. (In my opinion the cytologic formula has no bearing in these cases, since in the ball-valve, smooth-surface tumor cases it is always wanting.)

Third: Cases in which there is a variation in the coagulation. This may be temporary in the cases showing massive coagulation at times, or it may occur in cases which never show massive coagulation. Most of the imperfect cases belong to this group.

Of the cases showing meningitis and meningomyelitis, Mestrezat reports three cases:

CASE 1.—That of De Buch, in 1905, reported in the Congrès de Bruxelles. This was a case of transverse myelitis with spontaneous coagulation and fibrin and containing 0 5 per cent. of albumin.

CASE 2.—The case of Renon, Monier and Vinard. This was reported in *Soc. méd. des hôp.* in July, 1909. This case showed an ascending paralysis of the Landry type with cure. It contained also abundant fibrin and albumin.

Case 3.—The case of Froin's, reported in *Gaz. de hôp.*, November 12, 1905. This was a hemorrhagic fibrinous tuberculous meningitis with abundant albumin, a spontaneous coagulum, and a very notable xanthochromia. There was no coagulation *en masse.* The autopsy showed a tuberculous meningitis with hemorrhage.

Of the cases showing sarcoma of the meninges, there are six on record:

Case 4.—That of Sicard, in 1902, reported in the thesis of Vigneras, in 1908. This was a case of spinal compression by a tumor which developed on the posterior surface of the vertebral column. The cerebrospinal fluid showed xanthochromia.

Case 5.—The case by Sicard (1902), published in the same thesis. A case of compression by a very vascular psammoma. The cerebrospinal fluid in this case was a greenish-yellow.

Case 6.—The case by Dufour (1904), referred to by Mestrezat. A diffuse sarcomatous meningitis of the spinal cord. The cerebrospinal fluid showed xanthochromia, with the presence of fibrin, but no massive coagulation.

Case 7.—The case of Walter Rindfleisch (1904), already referred to, and included in the true cases, as Case 26. This was a case of diffuse sarcomata of the meninges. The cerebrospinal fluid was a yellowish-brown, and the coagulum of fibrin was quite thick after a few hours. It contained 2.4 per cent. of albumin.

Case 8.—The case of Kawashima, published in 1910 in the *Arch. f. path. Anat. u. Hyg.*, 1910, vol. cci, p. 297. This showed xanthochromia without spontaneous coagulation and the presence of red blood-cells and lymphocytes.

Case 9.—Case of Mestrezat, published in his book. This was the case of a patient who had fallen from a ladder. The cerebrospinal fluid showed a yellowish color without massive coagulation, the clot of fibrin forming only after the addition of some blood-serum.

Besides these cases reported by Mestrezat, a large number of others have appeared in the French and German literature, also belonging probably to the incomplete cases. My own feeling is that in these incomplete cases there is no true pocketing off of the lumbar culdesac. I feel they do not properly belong in the syndrome at all. Possibly they may at some future time be correlated with the true cases falling in the syndrome, and be found to be caused by transitory or evanescent adhesions or mere agglutinations; or by compression of an annular sort of the spinal canal, of varying degree, only occasionally reaching that required for the establishment of the complete syndrome.

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COMMENTS AND OPERATION

Dr. Murphy: The patient's history gives no suggestion of the existence of venereal infection; but he suffered a trauma twenty years ago. Perhaps there may have been some connection between that injury and his present complaints. At any rate, fifteen years ago he had some sort of a disturbance in one of his limbs, accompanied by pain, and a second, somewhat similar attack some years later. Two years ago, that is, in 1913, he began to have these pains in the lower limbs more frequently. The pains radiated down the limbs more posteriorly than anteriorly, apparently in the region of distribution of the sciatic nerve. Sometimes they would be more severe in one spot, and sometimes in another; but they have always been located below a line which would correspond approximately with the iliohypogastric nerve; they have never occurred above the level of the line joining the two anterior-superior spines.

Following the first spinal puncture mentioned in Dr. Mix's account of the case, there was a decided improvement in the patient's condition; that is, his pain became less and he felt decidedly better generally. The fluid, when withdrawn, was yellow in color and coagulated almost at once. It was this spontaneous coagulation particularly which first led us to consider the possibility of the existence of a circumscribed pathologic lesion of the lower cord or cauda, because normal spinal fluid does not coagulate until after long standing, if at all. That single phenomenon is what started us on this special investigation of the syndrome consisting of xanthochromia, massive coagulation, and lymphocytosis of the spinal fluid.

At the second aspiration the fluid coagulated in the same way, but was somewhat slower in its clotting, and a smaller number of white cells was found; but again the patient was relieved of his pain.

Dr. Mix has searched the literature of the subject carefully and has found, chiefly in the French literature, certain other cases where the spinal fluid coagulated spontaneously. These cases have usually shown immediate spontaneous coagulation *en masse* and lymphocytosis of the spinal fluid, and almost constantly a yellow coloration of the fluid—the so-called xanthochromia. Those cases which have come to operation or autopsy have shown a circumscribed annular constriction of the spinal meningeal space, due either to meningitis of low virulence, to tumor, or to external compression. This constriction in all the cases apparently has been sufficient to divide the meningeal space into two cavities, one above and one below, each distinctly separate from the other. The normal circulation of the spinal fluid is thus cut off. The constricting lesion, by its annular compression of the cord or cauda, may also shut off the nerve-supply to the regions below, producing sensory disturbances, sphincter disturbances, and pareses or paralyses.

Up to the present time Dr. Mix has collected 34 cases of this syndrome; in 11 of them death occurred. Four cases have been operated on, excluding our present case, and all so far have recovered. Of the non-operated cases, 8 died before the publications containing the report of them appeared. How many may have died since the reports as the result of the affection we do not know.

Considering the high mortality that has occurred in these cases, when allowed to pursue their natural course, and considering the very favorable results which have followed operative intervention, we consider it quite probable that this man will die if we do not relieve the lesion occluding his meningeal canal about the upper level of the conus; and if we *do* relieve this constriction by an operative procedure, the prospects of securing an ultimately good result for him seem very bright.

We have an operative procedure (described in the CLINICS, February, 1914, p. 166 *et seq.*) by which we can relieve this patient's symptoms if they are caused by tumor, even though we be unable to remove the tumor *in toto*. This operation is a decompression of the cord, similar in principle to cerebral decompression, and even more efficacious in its action. We have already used it for the relief of gummata and osteomata pressing on the cord, and, more frequently, for inoperable tuberculomata of the cord.

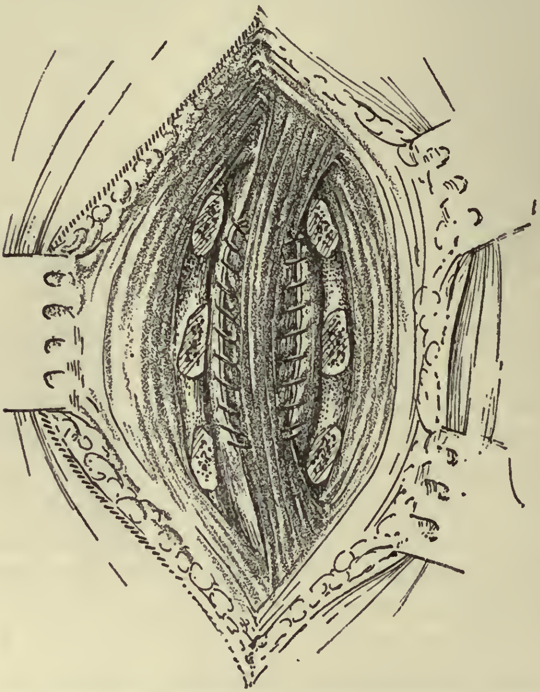


Fig. 84.—Dr. Murphy's operation for the relief of chronic obliterative meningomyelitis associated with sac-formation below the zone of constriction. (See Clinics for February, 1914.) It is this condition which most frequently gives rise to the syndrome consisting of xanthochromia, coagulation *en masse*, and lymphocytosis of the spinal fluid. Dr. Murphy's operation consists in the excision of the constricting membrane, with or without the dura, as necessary, and transplantation into the defect of either a strip of the lumbar fascia or of the erector spinæ muscle. This transplanted strip prevents the leakage of cerebrospinal fluid and reëstablishes the continuity of the meningeal canal. Dr. Murphy prefers the muscle transplantation which is here depicted. Where excision of the membrane is not practicable, Dr. Murphy would use Handley's tubular silk to reëstablish communication between the two sacs. Cerebrospinal fluid would seep along the silk, just as lymph does when the silk is used for the relief of the lymph stasis of elephantiasis.

If a tumor can be removed readily, the usual operative procedure is simple, radical, and the result very satisfactory. When, however, a tuberculoma, osteoma, or other bone tumor compresses the cord from in front, usually the pathologic lesion itself cannot be removed. Then we simply relieve the cord of the pressure by removing the posterior arch of the spinal canal, consisting of the spinous processes and laminæ. If necessary to relieve an excess of tension, we also divide the dura and implant in the defect a pedicled strip of lumbar fascia, suturing it between the edges of the split dura. Of course, this operation must be performed before the pressure on the cord has become sufficient to produce destruction or degeneration of the latter. The lumbar fascia, or perhaps, even still better, a strip of muscle tissue from the erector spinæ, will gradually stretch if the tumor continues to grow, and so for a long time one may prevent the development of sufficient intraspinal tension to destroy the conduction of nerve impulses in the cord. All our cases so treated have healed by primary intention in spite of the presence of this strip of interposed material.

If I recall the literature correctly, this patient is the oldest in whom this syndrome has ever been recognized. Previous to this case, the oldest reported case was sixty-seven years of age. This patient is sixty-eight years old.

For me, one of the most striking features of the clinical aspect of this case has been the immediate great relief which, in both instances, followed the aspiration of the fluid from the lower meningeal sac, and the almost machine-like gradual return of the pain and other nerve-root symptoms, as the occluded portion of the meningeal canal gradually refilled with the fluid.

[Dr. Murphy made the usual dorsal median incision, extending from the eleventh dorsal spinous process above to the spinous process of the fourth lumbar vertebra below.]

The tip of the conus usually comes to the lower margin of the first lumbar vertebra; and since we know from our examination of the patient that his lesion lies in the conus, we shall, therefore, remove the dorsal covering of the cord over the eleventh and twelfth dorsal and first and second lumbar vertebræ. This should give us a complete exposure of the area of the cord involved in the pathologic process. I am making a slight cross-cut on the skin at the level of the spinous process of the first lumbar vertebra, to blaze it as the center of my field of operation, since it is at this level that the tip of the conus is to be found. As we dissect away the lumbar muscles from their attachments to the spinous processes and the interspinous ligaments, we are using towel pressure continuously to diminish the oozing from the muscles in order to save the patient all the blood we can. Loss of blood to an old man is always a relatively serious proposition, because his blood-vessels cannot adapt themselves readily to the contracted circulation, as do the elastic vessels of younger individuals. While working on one side of the spinous processes we pack our incision on the other side tightly with gauze, and press on the muscles through the skin with towels. This pressure stays the venous oozing, and, although the bleeding may be quite profuse at first, as it is here, it rapidly subsides after a few minutes of this compression; and the operation is not delayed, because we proceed with the separation of the muscles on the opposite side of the spinous processes.

In order to get at the laminæ more easily we shall first cut away the spinous processes with an obtuse-angled heavy bone-cutting forceps. The removal of the spinous processes gives us excellent access to the bony arch covering the spinal canal. We next insert the rongeur forceps at the lower angle of the wound, and rapidly bite out the laminæ, proceeding in an upward direction, first on one side and then on the other. We cut the laminæ completely away on each side, so as to leave a space of maximum breadth between the bone edges.

On palpating the cord through the dura I find that there is an area of increased resistance present, resembling a tumor and extending downward almost to the lower limit of the exposed dura, and upward to the level of the lower margin of the body of the eleventh dorsal vertebra. Our compression has now stopped all the muscular bleeding, and we are preparing to open the dura, beginning the incision at the lower angle of the wound. We split the dura the full length of its exposure in the field. As the edges of the incision spread apart one can now see a mass appear-

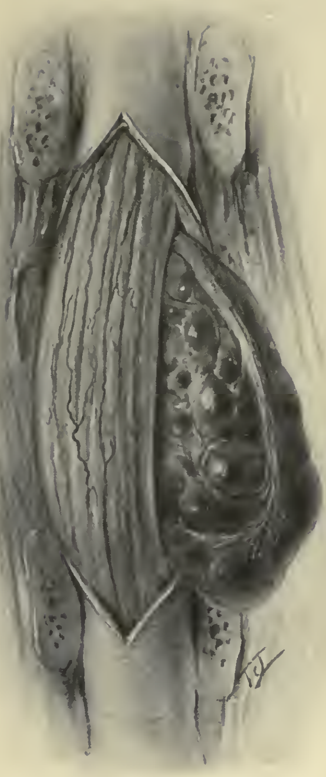


Fig. 85.—This natural size drawing shows the tumor as it spontaneously extruded itself between the fasciculi of the cauda out through the dural incision.

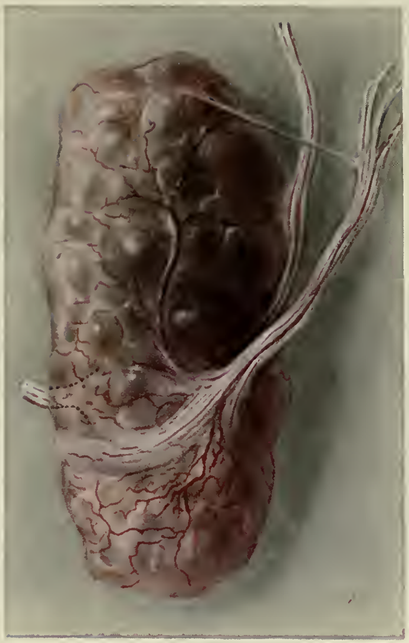


Fig. 86.—Intradural cystic tumor involving the filaments of the cauda equina. Drawing in natural colors, 1½ times natural size. Note the intimate connection of the filaments of the cauda with the capsule of the tumor.

ing in the field, apparently a tumor of some sort, which is being slowly extruded upward between the filaments of the cauda and out through the dural incision by the pressure from within the spinal canal.

Those who were close at hand noticed when I first inserted my scissors to divide the dura that a yellow fluid escaped. Then, when my scissors reached the upper end of the dural incision, there was another gush of fluid, watery in appearance, and perfectly colorless—apparently normal spinal fluid under somewhat increased pressure.

While we have been waiting a few minutes and talking, the tumor has been protruding itself still further. It also seems to have grown somewhat larger, and now appears to measure an inch and a half or two inches in length. It also is changing its shape a little as it emerges, and is becoming more conical and less oblong. Those who are near at hand may wonder how so large a tumor could be contained within the relatively narrow spinal canal. Of course, when lying in the canal it was compressed to a shape much more sausage-like and much less spheric than it is now.

I am rolling the tumor from side to side to see if I can free it easily from its surroundings. It appears to have two attachments—one above and one below. These attachments are not heavy and appear to consist of simply a single fasciculus of the cauda, the fasciculus entering the tumor at its upper end, and emerging from it at its lower end. From all other attachments I can separate the growth readily. Apparently, therefore, we must cut this caudal filament above and below, to free the tumor *in toto*. This excision might not be absolutely necessary if we could be certain that the tumor is benign; but since many of these meningeal tumors are malignant in character, it will be safer to remove this single filament, even though it should cause some slight functional defect, than to take the chance of leaving a structure which may be infiltrated by malignant cells.

We have now removed the tumor completely and it appears to be an elliptic, translucent, pinkish, cystic tumor. We shall send this specimen to our pathologist at once for a thorough examination.

[The pathology of the case, as reported by the pathologist, Dr. W. O. Sweek, is as follows: On gross section the tumor shows numerous cysts, varying in size from those just visible to the eye up to those 5 mm. in diameter. These cysts in some instances are filled with blood; in others, with a colloid-like material.

On microscopic examination the tumor is seen to be composed of glial tissue, well supplied with vessels, and in spots undergoing degeneration. The larger cysts are filled with colloid material, which takes the eosin stain very deeply, and the other cysts are filled chiefly with blood-cells. The vessel-walls are thick and in some instances show a marked inflammatory reaction. In several areas of the tumor there is a round-cell infiltration. The tumor, therefore, belongs in the group of cystic gliomas.]

Dr. Murphy: In these cases of spinal tumor it is not at all uncommon to have the paraplegia appear very suddenly, notwithstanding that the tumor may already have been growing for a considerable period of time. In one case of ours, a metastatic sarcoma of the spinal cord at the level of the fourth dorsal vertebra, we demonstrated the patient at a Saturday clinic as a case of complete recovery from a sarcoma of the scapula treated by radical scapular excision. That was two years and eight months after the excision. Only four days later, on a Wednesday, she developed some pains in the lower limbs, and within two hours after the onset of the pain became completely paraplegic. I thought at first that she had suffered a hemorrhage into the cord. We operated almost immediately, and found a central sarcoma of the spinal cord of the same cell type as the original tumor, filling the spinal canal and infiltrating the cord. The case went rapidly on to a fatal termination. I cite this patient merely to show that entire freedom from symptoms may exist in these cases of tumor of the spinal cord for a long period of time, and that when paralysis does set in, its onset may be very sudden.

The plan of treatment which we had outlined for this case,

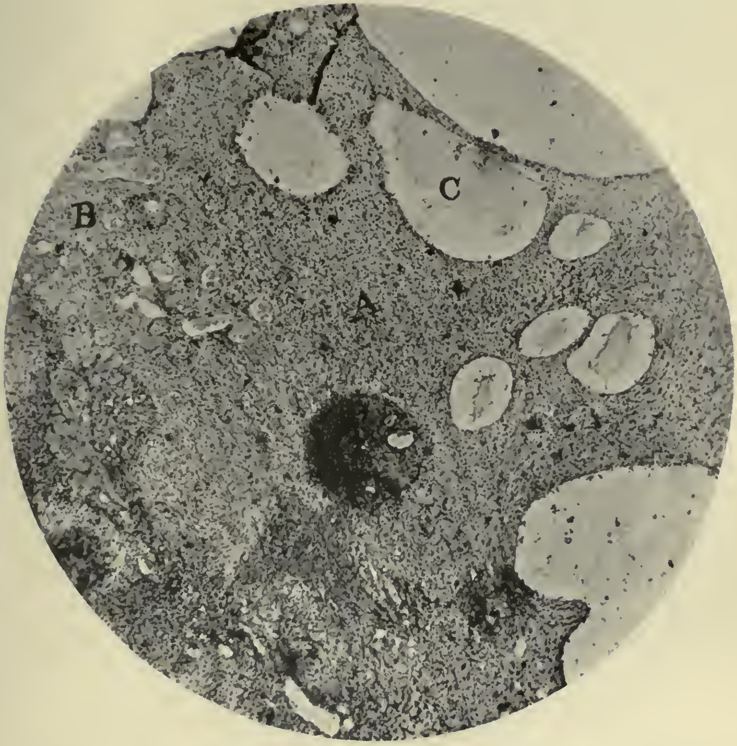


Fig. 87.—Cystic glioma of the spinal cord. The solid portions of the tumor seem to be made up of—*A*, Glia-like cells interspersed with numerous thin-walled blood-vessels, *B*, and containing many cysts, the contents of which are chiefly colloid, *C* (magnification, 46 diameters).

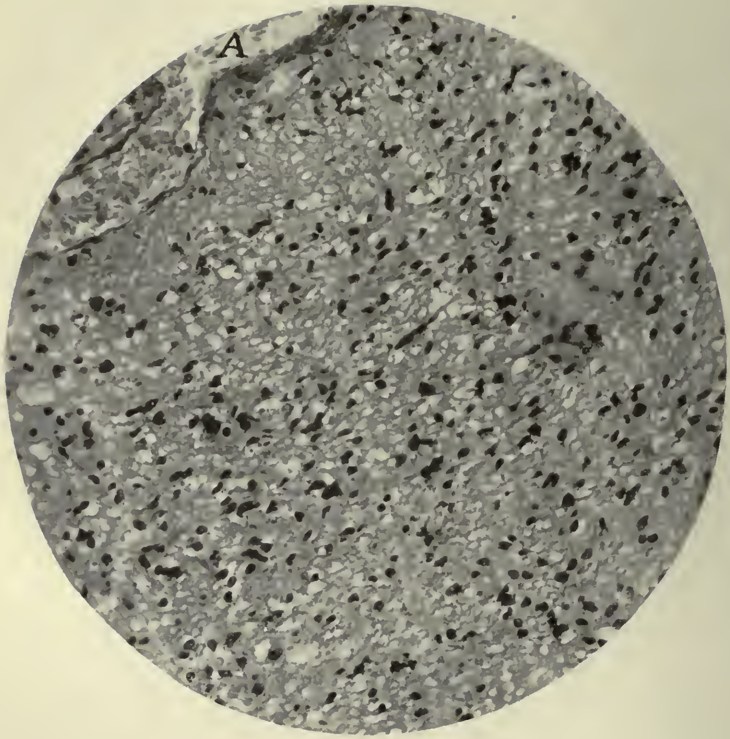


Fig. 88.—Cystic glioma of the spinal cord. This field shows an area in the cellular infiltrate with a blood capillary, *A*, at the extreme edge of the field. Note that the wall of the capillary is made up of endothelial cells and not of tumor tissue, as is frequently the case in sarcoma (magnification, 230 diameters).

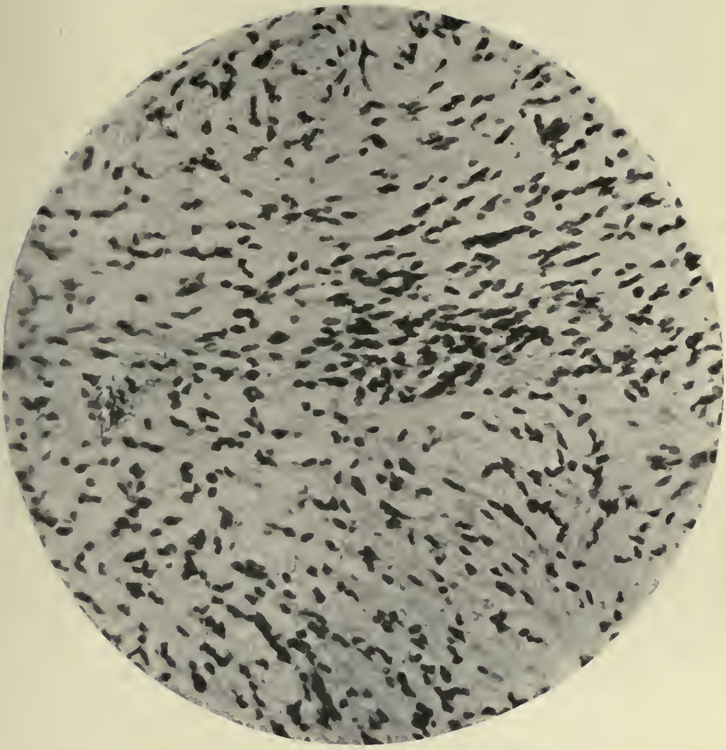


Fig. 89.—Cystic glioma of the spinal cord. Another field of the cellular infiltrate showing some tendency to grouping into columns and whorls (magnification, 230 diameters).

if it had proved to be a circumscribed meningeal inflammatory process too intimately connected with the cord and cauda to permit of excision, was to use Handley's tubular silk, passing it through the dura at a level above the old inflammatory process, across through the focus itself, and out of the dura below the lesion, that is, clear through the lumbar culdesac, so as to reëstablish communication between the upper and lower cavities, with the hope that the spinal fluid might follow this path again in its circulation.

Handley originally used this tubular silk to maintain the lymphatic circulation in cases of elephantiasis, particularly elephantiasis of the legs. The silk strands were inserted just beneath the skin; the lymph circulation then followed the paths thus opened up and maintained, and the lymphedema of the limbs was correspondingly diminished.

In Dr. Mix's report of the literature of this subject some of the cases were apparently due to a spinal meningitis caused by various intradural injections, even when the injection material, on *a priori* grounds, would appear to have been of an innocuous nature. One would hardly have expected stovain, for instance, to have produced so violent an inflammatory reaction in the glial connective tissue as it apparently did in one case. The moral to be drawn from these injection-meningitis cases is: *Avoid intraspinal injections!* Those who recommend the extensive use of intraspinal anesthesia should not disregard these rather frequent evil results.

Since we have been able to remove this tumor *in toto* we should secure the patient a good result. Apparently, judging by the results of the cases previously operated, as well as by this case, this operation should open up a new and promising, even if rather limited, field of work.

Eight of the reported cases were definitely tumors. Two cases were neither operated nor autopsied; and, although suspected to be tumors from their symptomatology, they were not definitely proved to be such, either by operation or by autopsy. Of the 34 cases, 24 were due to circumscribed meningitis, either in the caudal region or in the region of the conus or the lower lumbar cord. Only 2 of the cases appeared to have lesions distinctly higher up than the conus.

How many of the cases were operable and how many inoperable? That we cannot state. None of them were operated, and in none of them was operation suggested. Seven or 8 of them apparently partially or completely recovered under medical treatment; the others went on either to a fatal termination or were ultimately lost sight of before death occurred. In view of the very successful results following operation in these cases, and the very dubious prognosis of the unoperated cases, it would rather seem that operative procedures should be resorted to much more frequently than has been the case in the past. Certainly all cases of this group which do not promptly improve under medical treatment should have the benefit of an operative procedure which offers them an excellent prospect of complete cure.

[Note.—The patient had some pain the night following the operation. In seventy-two hours all his pain had disappeared, and we were able to discontinue the injections of morphin which he had been taking in large doses for a couple of years. Sensation and motion rapidly returned to normal in the lower extremities, and five weeks after the operation the patient was permitted to return home. His course since he left the hospital has been an uninterrupted one to a complete recovery.—Ed.]

RICHMOND STEPHENS, M. D.

Volume IV
THE CLINICS
OF
JOHN B. MURPHY
Number 3

MURPHY'S CLINICAL TALKS ON SURGICAL AND GENERAL DIAGNOSIS

A CLINICAL TALK ON THE DIAGNOSIS OF INJURIES OF THE CARPUS

Dr. Murphy: We have had a number of cases of injuries of the carpus recently, and since this subject is clinically important, as well as very interesting, I want to lay stress on some of its important aspects, which our cases illustrate.

The *x*-ray has been a great revelation to us in the matter of bone pathology. Formerly we knew only the dead-house aspects of bone pathology, or had fragmentary glimpses of living pathology on the operating-table. Now, with the *x*-ray, we are able to follow the pathologic changes in bone from day to day with great thoroughness and in minute detail. So accurate is our knowledge of the gross pathologic changes of bones becoming that we are depending more and more on the clinical aspects of the cases and the *x*-ray plates, and less and less on microscopic examination.

Rovsing, of Copenhagen, stated a few years ago that he preferred an *x*-ray plate to a microscopic section in making a diagnosis of most bone lesions. That statement represents the standpoint of modern surgical practice to-day. If anything, I believe it underrates, rather than overestimates, the value of the *x*-ray. If I were formulating an opinion, I think that I should put it this way: In practically all bone lesions an *x*-ray examination is essential to an accurate diagnosis; when the proper *x*-ray examination has been carried out, a histologic study of the lesion will yield additional important diagnostic evidence only in the exceptional case.

This statement is made with particular reference to inflammations and tumors of bone. In osteal injuries one does not expect, and never has expected, the microscope to yield any very important evidence. In the diagnosis of injuries of the bones the use of the x-ray, however, has made possible just as many advances in our knowledge as it has in the case of bone tumors and inflammations, and perhaps even more. The use of the x-ray has, in fact, revolutionized our ideas on the subject of fractures and dislocations. We can diagnose fractures, individualize in their treatment, and control our results with an accuracy undreamed of in the pre-Roentgen era.

Before the x-ray was discovered most surgeons looked upon fractures and dislocations as a subject pretty well worked out and exhausted—a relatively fruitless field for research. But since the x-ray tube became a clinical instrument, the advances in this field of surgery have been so rapid and numerous that no surgical text-book has been able to keep abreast of them. In fact, there exists no text-book which adequately represents modern ideas on fractures and dislocations, and even if such a text-book were started now, by the time it was written and published it would no longer be strictly up-to-date.

A great mass of careful x-ray observations of the various fractures and dislocations is already in existence. What we now need are adequate studies of individual lesions and certain groups of lesions, using this immense accumulated material as a basis. Quénu, of Paris, has made a study like this of fractures about the ankle—a series of papers in the French *Archives de Chirurgie* for 1913, comprising several hundred pages of that publication. This is an excellent and comprehensive clinical study along the lines most needed at present.

A similar monograph on lesions about the wrist-joint would be equally welcome; and of this subject carpal fractures and dislocations make up an interesting and nearly brand-new chapter.

Very little was known of lesions of the carpus before the Roentgen era. Some of the investigators who did experimental fracture work on cadavers noticed, in the production of traumatic lesions about the wrist-joint, an occasional fracture or dislocation of one of the carpal bones, but attached no particular practical importance to the observation. Carpal lesions by most surgeons were presumed to be practically always the result of severe direct violence, resulting in the production of open wounds.

It is only since we came to use the *x*-ray systematically in the examination of injuries about the wrist-joint that we have realized the frequency of subcutaneous fractures and dislocations of the carpal bones. Little by little an enormous number of cases has been collected, and we have learned to recognize a considerable number, not only of typical, but also of atypical, lesions. For practical purposes, however, we have to consider only three lesions, and these are: First and most frequent, the fracture of the scaphoid bone; second, the dislocation, so called, of the semilunar, and, third, the combination of the two, scaphoid fracture plus semilunar dislocation. Of atypical injuries there is a great variety, but because of their infrequence they are only of minor importance. Even the fracture of the semilunar, which is the lesion next in frequency, is a comparative rarity. Since one of the cases I am showing you to-day is a scaphoid fracture, and since this is the most frequent carpal lesion, I shall mention some of the clinical characteristics of this fracture before taking up the case itself in detail.

SCAPHOID FRACTURE

We now know that fractures of the scaphoid bone make up at least 0.5 per cent. of all fractures in the body. All modern fracture statistics grant at least this degree of frequency. That fact gives this fracture an enormous importance, which, previous to the Roentgen era, we never should have suspected. The typical Colles' fracture of the lower end of the radius—the most frequent fracture in the body—makes up only 10 per cent. of all fractures; and realizing that fact one can understand the urgent necessity of carefully examining the *x*-ray plates of every injured wrist for the possible presence of this fracture. From my own experience I should say that this percentage (0.5 per cent.) is too low, and would be much more inclined to estimate the frequency of scaphoid fractures at from 1 to 2 per cent. of all fractures. In other words, I think that, in my experience, I have found about one fracture of the scaphoid to every ten or less Colles' fractures.

Why does the scaphoid so often suffer in injuries of the wrist? When one stops to figure out the reason, the mechanism at once becomes evident. The scaphoid lies interposed between the lower end of the radius and the distal row of carpal bones. Its position with relation to this distal row and also to the radius depends upon the position of the hand. In outward, that is, in radial, flexion of the hand the scaphoid comes to lie between the lower end of the radius and the os magnum. Force transmitted upward from the palm of the hand will jam the scaphoid, under such circumstances, between these two bones, both of which often are more solid in their structure than the scaphoid itself. Furthermore, the concave surface of the scaphoid receives the os magnum like a wedge, and this wedge action breaks the scaphoid when exerted at the latter's weakest spot, its concave middle. Force directed downward from the radius acts in exactly the same way, making the scaphoid the buffer in both these classes of injuries. When, however, the hand is in ulnar or inward flexion, the scaphoid lies further outward, between the styloid process of the radius and the trapezium and trapezoid. In this position it is less exposed to fracture, since its long axis is presented to the acting force; but, nevertheless, in some cases an impacted fracture may result, and in other cases, where the hand is thrown over violently into ulnar flexion, the external radioscaphoid ligament occasionally tears off the tuberosity of the scaphoid, the ligament being stronger often than the bone itself.

There are, therefore, three typical fractures of the scaphoid —the simple transverse fracture; the transverse fracture with impaction, which usually later results in bone absorption at the line of fracture, and apparent cavity formation; and the fracture of the tuberosity.

The etiology of these fractures is very similar to that of a Colles' fracture. They are produced by falls on the hand, by back-firing of an automobile while cranking, or by direct violence. By looking at the *x*-ray plate one can usually tell just about how the accident happened; and if the patient gives an accurate

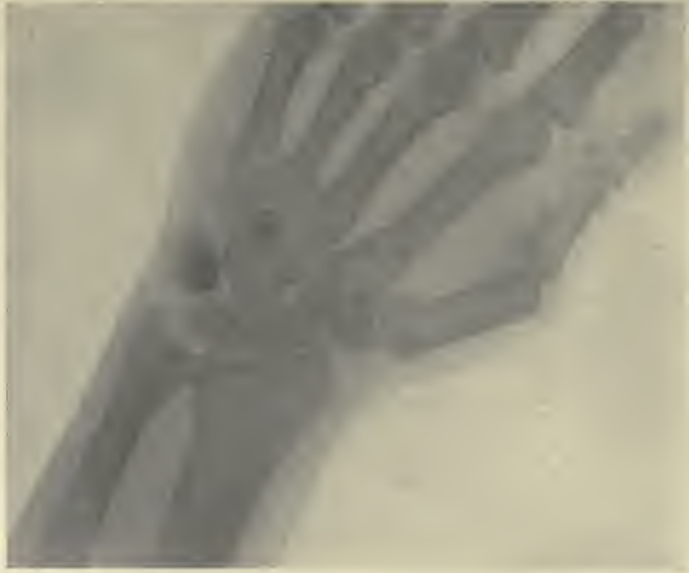


Fig. 90.—Normal hand in radial flexion. Note that in this position the scaphoid is interposed over its entire extent between the radius and the distal row of carpal bones, and that it must transfer to the radius any force applied to the palm in an upward direction, and vice versa. Hence fracture of the scaphoid usually occurs when the hand is in radial flexion. Note that in this position the semilunar bone lies practically outside the danger zone, far enough to the ulnar side to escape the maximum violence of any trauma acting on the palm of the hand in an upward direction.

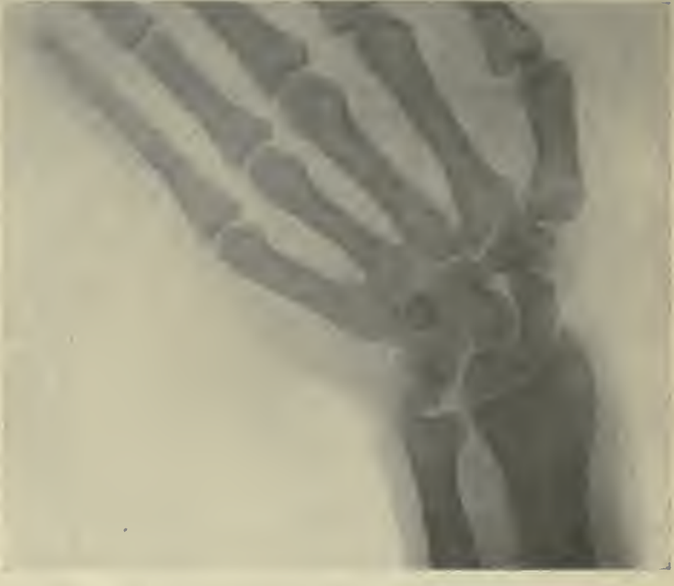


Fig. 91.—Normal hand in ulnar flexion. Note that in this position the greater part of the scaphoid has escaped from the concavity of the articular surface of the radius, and that only its most proximal portion, together with the semilunar bone, now lies interposed between the radial head and the distal row of carpal bones, especially the os magnum. The latter fits into the concavity of the semilunar and adjacent portion of the scaphoid like a wedge. It is in this position that the semilunar bone suffers most when exposed to violence applied to the palm, and that semilunar fracture most often occurs. Still more frequently semilunar dislocation either with, or more often without, an accompanying transverse fracture through the body of the scaphoid takes place.

history as to how he fell, one can sometimes prophesy just what the x-ray plate will show.

The x-ray has made the diagnosis of these fractures very simple, but at the same time it has added so greatly to the confirmation of their symptomatology that we are now able in most cases to make a fairly positive diagnosis of them even before having recourse to the roentgenogram. In the first place, the history is very suggestive. The patient falls on his palm and tells you, as a rule, that his fingers were turned toward him—that is, his hand was in radial flexion, as he fell, because this is the position in which the scaphoid is most exposed to the wedge action of the os magnum. On the other hand, impacted fractures more often are cranking fractures, because these are obtained, as a rule, when the hand is in the position of ulnar flexion. Tearing off of the tuberosity is also most frequently caused by cranking or by some other sudden ulnar flexion of the hand.

In carrying out the examination of such a case one does not first examine the carpus. The first step necessary is to assure oneself that the radius and ulna are intact, and particularly that no impacted fracture of the former exists, and one does this by demonstrating the absence of tenderness, crepitus, and deformity at the lower end of the radius, especially over the styloid process and at the lower end of the ulna.

After thus eliminating the possibility of a fracture of the forearm bones, and coming to the examination of the carpus, one will probably note next that the swelling and deformity lie distal to the line connecting the two styloid processes, and that observation at once suggests a carpal injury. When, therefore, a carpal injury is suspected, one next examines the scaphoid, since that is the bone most frequently injured by carpal violence. First palpate the "anatomic snuff-box." This is the depression at the base of the thumb, at the outer side of the carpus, and lies between the extensor pollicis longus on one side and the extensor pollicis brevis and abductor pollicis longus on the other side. In examining the "anatomic snuff-box" to determine the presence of a scaphoid fracture it is best to have the patient's hand held in ulnar flexion, because that position brings the scaphoid out from between the os magnum and the radius and into the "snuff-box." Tenderness is the symptom one hunts for here, and both in recent and old-standing injuries of the wrist this tenderness in the "snuff-box" is a very suggestive symptom. Besides tenderness, swelling in the "snuff-box" is usually also present,—always in acute lesions and frequently in chronic lesions,—being due in the latter event either to callus or joint effusion.

One next, probably, notices or is told by the patient that the movement of the wrist-joint is limited, both in flexion and extension. This limitation of motion is partly due to pain and partly to effusion into the wrist-joint, and, in old standing cases, partly to atrophy of the muscles about the joint and chronic arthritic changes and infiltrations.

Besides the two symptoms mentioned the other common signs of fracture may sometimes be elicited and sometimes not, such as crepitation, hemorrhage into the skin, and shortening of the carpus. The shortening of the carpus is not present in all cases, but is most often noted in impacted fractures. It is determined by measuring the distance between the tip of the styloid process of the radius and the base of the first metacarpal bone with a pair of draftsman's calipers.

The strength of the hand, as shown by the grip, is, of course, diminished, because of the pain which gripping produces, and, in chronic lesions, is diminished also by atrophy of the neighboring muscles.

Lastly, we come to a symptom which I have found of great value, and on which I think sufficient stress has not been laid by most surgeons; that is, the pain produced by jarring the scaphoid indirectly. I can make clear what I mean perhaps better by illustrating. Holding the hand in radial flexion so as to bring the scaphoid between the os magnum and the lower end of the radius, I have the patient flex his fingers, and then, with the percussion hammer, I gently strike the distal ends of the metacarpal bones, a method first demonstrated to me by my associate, Dr. Roger T. Vaughan, who has an emergency surgery service at the Cook County Hospital and often sees these cases.

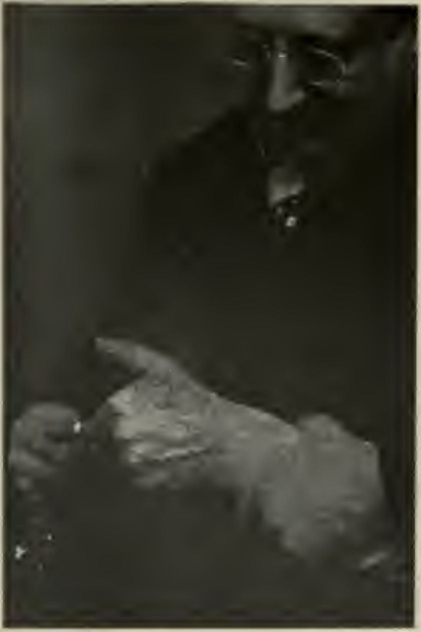


Fig. 92.—Correct method of performing the knuckle percussion test in diagnosing carpal injuries. The surgeon stands in front of the patient, grasping the latter's left wrist with his left (or vice versa), and holding it tightly against his body out of sight of the patient (Vaughan).

Percussion of middle knuckle in ulnar flexion: (*a*) Most painful with semilunar fracture. (*b*) Less painful with scaphoid fracture. (*c*) Still less painful with semilunar dislocation.

(Percussion over the ring finger with ulnar flexion of the hand is not painful at all in scaphoid fracture, but very painful in fracture of the semilunar.)

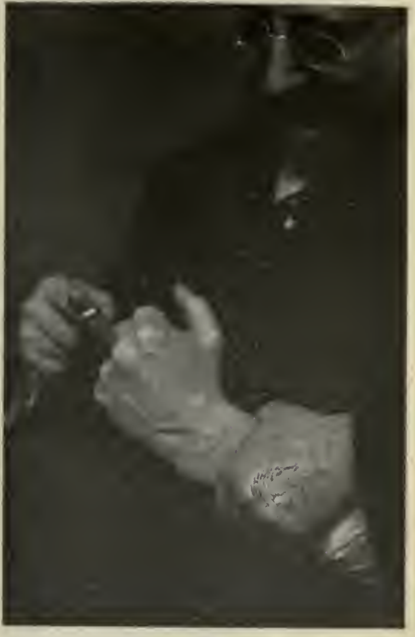


Fig. 93.—Correct method of performing the knuckle percussion test in diagnosing carpal injuries. The surgeon stands in front of the patient, grasping the latter's left wrist with his left (or vice versa) and holding it tightly against his body out of sight of the patient (Vaughan).

Percussion of middle knuckle in radial flexion: (*a*) Most painful in scaphoid fracture (but percussion over the knuckle of the index-finger is still more painful and over the ring-finger knuckle not painful at all). (*b*) Very painful with semilunar fracture. (*c*) Not painful with semilunar dislocation (any to speak of).

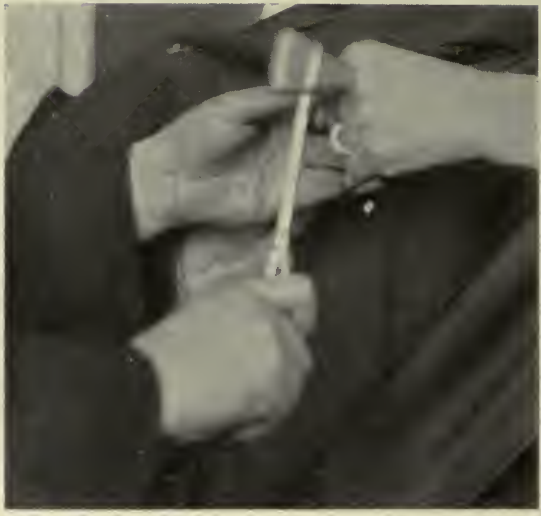


Fig. 94.—Showing how *not* to perform the knuckle percussion test for eliciting tenderness with fracture of the scaphoid and fracture of the semilunar. Unless the surgeon has firm control over the patient's hand and wrist, the latter is likely to wince before the knuckle is struck and so destroy the accuracy of the test. It is also well to have the patient close his eyes or look the other way before the test is made.

In striking the third metacarpal the os magnum is driven against the scaphoid, and in a case of scaphoid fracture even a very slight tap causes intense pain. Not only does tapping the head of the third metacarpal cause this symptom, but tapping of the first and second metacarpals, which transmit the impulse to the scaphoid by way of the trapezium and trapezoid, also produces marked pain. The fourth and fifth metacarpals, however, are insensitive when tapped, and thus a fracture of the scaphoid may be differentiated from one of the semilunar. Furthermore, the "tapping-pain" in scaphoid fracture is most marked in radial flexion, while in semilunar fracture the pain is most marked in ulnar flexion.

We next come to our final and most positive means of diagnosis, the *x*-ray plate. If the *x*-ray plate be not carefully made, and if the bone detail is not clearly evident in it, a fracture may be overlooked in the plate which the clinical signs and symptoms make fairly evident. The explanation is as follows: The common fracture through the body of the scaphoid is a mere transverse split without displacement, and the fragments subsequent to fracture may be so accurately readjusted that the line of fracture is represented by a thin line which can be seen only on an *x*-ray plate showing sharp bone details. One can, therefore, readily see how important it is to recognize the frequency of this fracture, and to possess a knowledge of its clinical characteristics, if one wishes to avoid the grave error of overlooking its presence. For if a fractured scaphoid be overlooked and the patient told that he has only a sprain of the wrist, and ordered to bathe it, to exercise it, and to go back to his work, little by little the disability of the wrist will increase, chronic arthritic changes will set in as the result of the chronic irritation of the joint, the muscles about the joint will slowly atrophy, and the man will lose the use of what should have become a useful hand.

All these evil sequelæ develop because, as a rule, scaphoid fractures do not unite. The reason for this common failure of union is simple: The location of the scaphoid is almost completely intra-articular; therefore its blood-supply is scanty and enters the bone by way of the neighboring ligaments. When the bone is fractured at the usual site, the nutrient vessels which enter it near its middle, that is, at the site of the injury, are generally destroyed, and the source of the bone's nutrition is thus cut off. If the bone fragments do not actually become necrotic, at least their circulation is not sufficient to enable them to throw out callus. Then a pseudarthrosis may develop, but no union. This slight osteogenetic power of the scaphoid is quite like that of the sesamoid bones, which usually fail to unite when fractured, their blood-supply also being sparse and entering them through dense ligaments. This is almost invariably what happens in intra-articular scaphoid fractures. The fracture of the tuberosity of the scaphoid is the only exception to this rule. Here the line of fracture lies outside the joint, and the blood-supply to both fragments is preserved; therefore union practically always occurs if assisted by proper immobilization.

What, then, are our indications for treatment? In fractures of the body the dead fragments of the scaphoid in many instances act exactly like foreign bodies in the joint. They must be removed. On the other hand, fractures through the tuberosity have no such untoward action on the joint, and heal readily under immobilizing treatment. In the ordinary fracture of the scaphoid, therefore, where the pseudo-arthrosis present causes continued pain and disability, the only point to consider is *how* we shall operate—whether through a dorsal incision, whether through a palmar incision, and whether these incisions shall be longitudinal or transverse. As a matter of fact, I think it does not make much difference which one uses, so long as one gains free access to the bone, avoids injuring the tendons and nerves, and is enabled so to reunite the skin as to avoid adhesions between the skin and the tendons. Personally, I have used most frequently the long palmar incision.

DISLOCATION OF THE SEMILUNAR

One cannot gain a proper understanding of the so-called *dislocation of the semilunar* and its variations without going into the entire subject of dislocations of the wrist, for semilunar dislocation is essentially a dorsal dislocation of the hand, and the most common variety of it.

The dislocations of the wrist which theoretically can occur are very different from those which actually do take place. On looking at the accompanying diagrams (Figs. 95, 96, 97), one notes that dislocation of the wrist apparently might occur: first, at the articular interval between the proximal line of carpal bones and the ulna and radius; second, it might occur at the intercarpal line, the line of articulation between the two carpal rows; third, it might take place between the distal row of carpals and the metacarpals. Although these three possibilities all exist, they are extremely rarely realized.

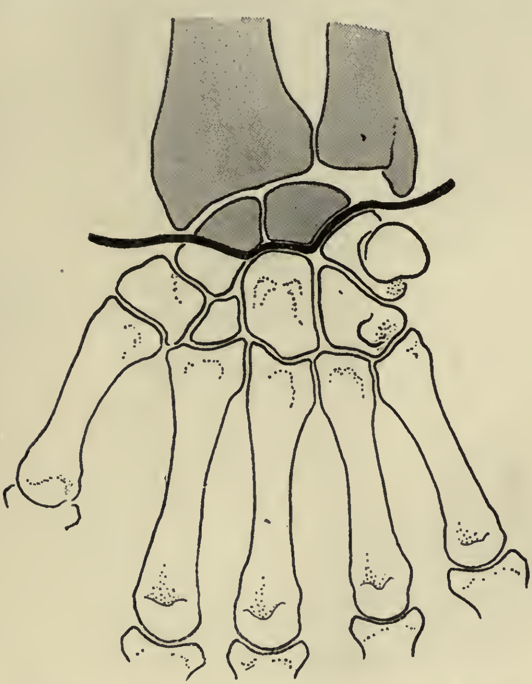


Fig. 95.—Dislocation of the wrist, showing the line of separation when the scaphoid is fractured and its ulnar half remains attached, with the semilunar bone, to the radius and ulna. (It is much more usual, when the line of division passes around the semilunar and through the scaphoid, for fracture of the ulnar styloid to occur than for rupture of the internal lateral ligament to take place.)

What actually happens is that the line of dislocation travels through the wrist, following the line of least resistance. This line of least resistance may pass partly through bone, partly through ligament, and partly through articular intervals. There are considerable individual variations in this line of combined dislocation and fracture. In some persons the ligaments are stronger and the bones weaker, and vice versa. The accompanying diagrams indicate most of the common variations. In spite of minor differences in anatomic behavior, these dislocations show a marked tendency to follow a certain line, and the diagrams well illustrate this point. Dislocation tends to occur proximal to the intercarpal articulation. The navicular and, much

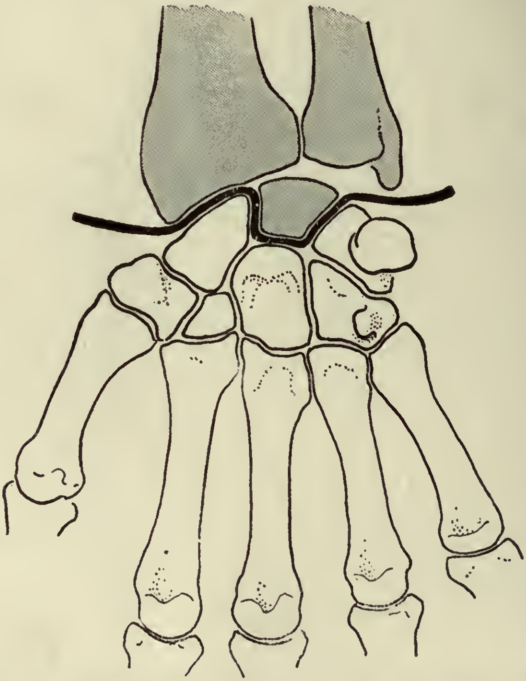


Fig. 96.—Dislocation of the wrist, showing the line of separation when the semilunar bone remains attached to the radius and ulna—the so-called dislocation of the semilunar. (It is much more common for the ulnar styloid to be broken off than it is for the internal lateral ligament to be ruptured, as here indicated.)

less frequently, the scaphoid, show a marked tendency to follow the forearm bones, whereas the cuneiform, pisiform, and distal row of carpals nearly always follow the dislocated hand.

The most frequent luxation at the wrist is that which occurs at the proximal articular interval, the line of luxation passing *around* the semilunar bone. In the majority of these dislocations the ulnar styloid is broken. In the minority of the cases the internal lateral ligament is torn. The lesion produced depends on which is the stronger, the ligament or the bone. Similarly, the tip of the radius is sometimes broken off, though, as a rule, it is the external lateral ligament which gives way instead.

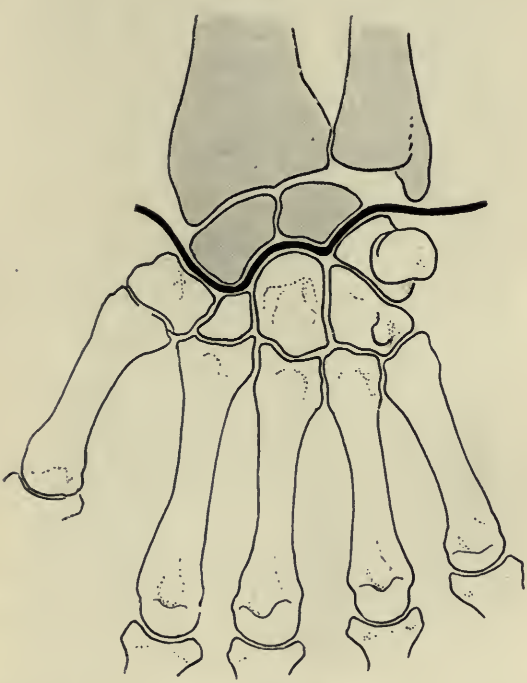


Fig. 97.—Dislocation of the wrist, showing the semilunar and scaphoid both remaining attached to the radius and ulna and the remaining portions of the carpus and the hand luxated. (It is much more usual for the line of division to pass *through* the scaphoid bone than around it.)

A rather common variation of this luxation is accompanied by fracture of the scaphoid through the neck, its weakest spot. Our own list shows three such cases, as against one without the scaphoid fracture; but statistics from the literature show that the isolated dislocation of the semilunar is more frequent than dislocation combined with scaphoid fracture. Occasionally the line of dislocation leaves the scaphoid and semilunar bones both clinging to the forearm bones; and, quite rarely, the cuneiform and pisiform may remain with the semilunar and the forearm bones and the scaphoid follow the hand. All these variations probably depend on anatomic variations in the hand as much as,

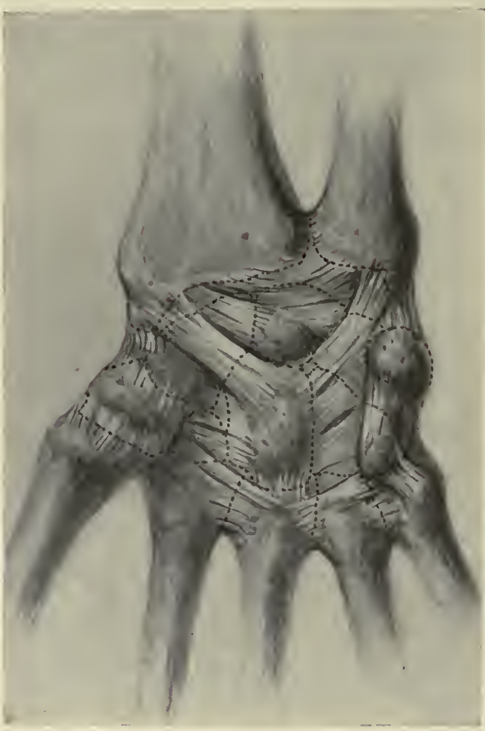


Fig. 98.—A drawing of the palmar carpal ligaments. The carpal bones are indicated in dotted outline. The results of trauma to the carpus depend largely on anatomic variations in the strength of these various ligaments, as well as of the bones. The ligaments play an especially important rôle in dislocations of the wrist, of which the so-called "dislocation of the semilunar bone" is the most frequent.

or even more than, they do on the character of the causative violence.

Why does the semilunar cling so closely to the forearm bones? It is because the anterior ligament binding it to the ulna and the radius, particularly the latter, is so much stronger than the ligamentous connections of the semilunar with the other bones of the carpus. (See the accompanying diagram, Fig. 100.) The anterior ligament is much stronger than the posterior ligament, and when the latter is torn, as it frequently is in these dislocations, the usual integrity of the anterior ligament produces a characteristic and progressive alteration in the location of the dislocated bone.

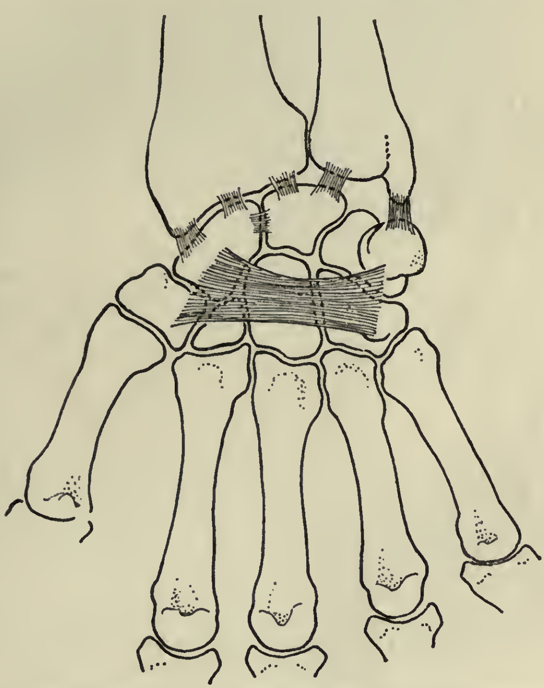
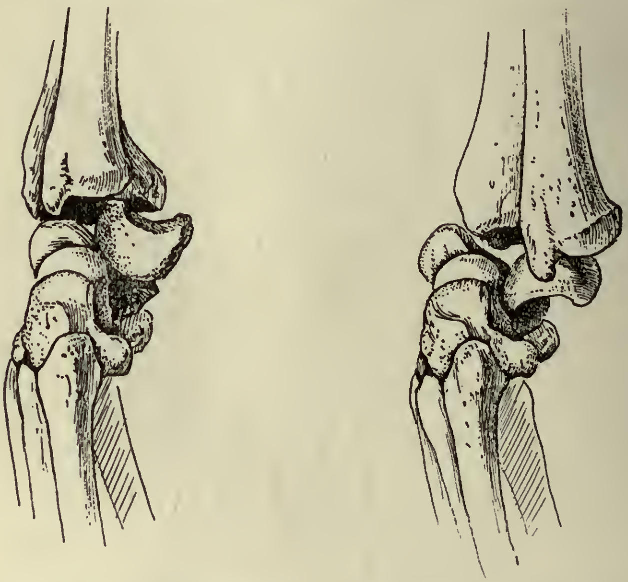


Fig. 99.—Diagrammatic drawing showing the essential ligamentous connections of the carpal bones. Especially tough and unyielding is the radio-ulnar ligament. Note the line of least resistance around the semilunar bone and through the neck of the scaphoid and the ulnar styloid. Compare this diagram with the accompanying drawing (Fig. 98) made from an actual dissection of the palmar carpal ligaments.

The first stage of this carpal lesion is the production of a dorsal dislocation of the hand on the wrist, with the semilunar bone remaining *in loco*, that is, fastened to the radius and ulna by the anterior ligament, and, less frequently, by the posterior also.

The second stage of the deformity is brought about only when the posterior radio-ulnar ligament has been ruptured, either by the original injury or by efforts at reduction of the luxation. The anterior pole of the semilunar then remaining fixed, the posterior pole rotates forward about the anterior pole as an axis, the degree of rotation depending on the mechanical factors present tending to pull the hand forward and upward.



Luxation of the semilunar bone forward, with rotation and without detachment from the radius—the third stage of semilunar dislocation. The second stage lies half-way between these two.

Backward luxation of the hand, with retention of the semilunar bone attached to the radius—the first stage of semilunar dislocation.

Fig. 100.—Dislocation of the semilunar bone.

So marked may this rotation become that the semilunar may

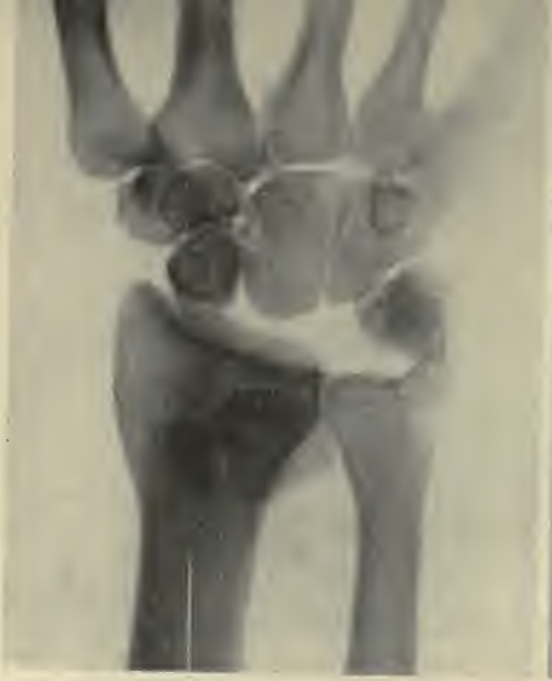


Fig. 101.—Transverse fracture of the scaphoid and dislocation of the semilunar. Postero-anterior roentgenogram before operation. This is the so-called "third stage" of semilunar dislocation, the semilunar bone having rotated forward and upward through an angle of 180 degrees. Its anterior horn is still attached to the radius by the anterior ligament, but the entire semilunar and the attached proximal fragment of the fractured scaphoid have come to lie completely proximal to the line of the wrist-joint. Note that, except for the defect due to the luxated bones, the appearance of the carpus is normal. The strong intercarpal ligaments maintain the bones in their usual relationships in spite of the defect in the row of proximal carpals.

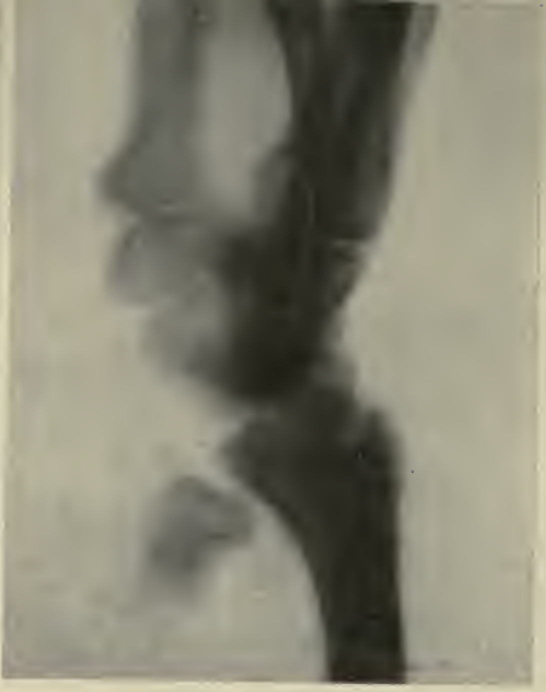


Fig. 102.—Transverse fracture of the scaphoid and dislocation of the semilunar (third stage). Lateral roentgenogram before operation. The details of this lateral roentgenogram are not sharp because the patient moved while it was being taken; but the position of the dislocated bones—on the anterior surface of the radius, entirely proximal to the wrist-joint—is unmistakable, and corroborate the conclusions drawn from the postero-anterior roentgenogram. (Case previously reported in the Clinics for June, 1913, p. 431.)

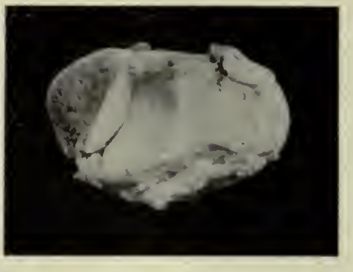


Fig. 103.—Transverse fracture of the scaphoid and dislocation of the semilunar. Photograph of the excised bones (distal surface). Note that the bones are relatively free from ligamentous attachments on the distal surface—that the ligaments lie chiefly on the anterior, posterior, and proximal surfaces.

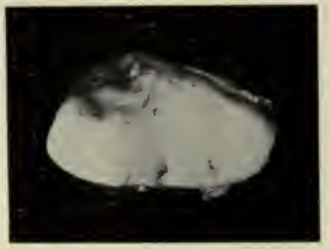


Fig. 104.—Transverse fracture of the scaphoid and dislocation of the semilunar. Photograph of the excised bones (proximal surface). Note the intimacy of their ligamentous connection.

turn through an arc of more than 180 degrees, and come to lie well up on the anterior surface of the forearm, in the so-called third stage of the affection. One of our cases had such a deformity. Not only the semilunar bone, but also the proximal half of the fractured scaphoid attached to the semilunar, passed up the anterior surface of the wrist and made a distinct and painful lump an inch above the articular interval of the wrist. (See Clinics for June, 1913.)

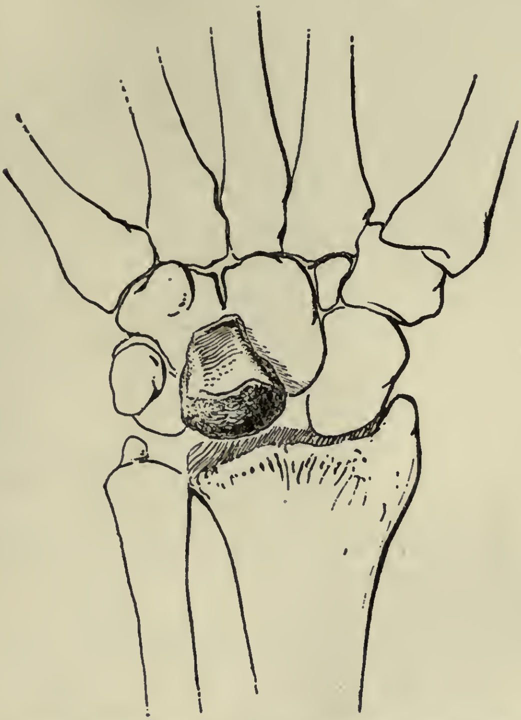


Fig. 105.—Outline of the semilunar bone in the first stage of dislocation. Compare the drawing of the skeleton hand with the drawing of the hand of the normal skeleton, and note the marked and characteristic difference in outline. Compare this drawing with the anteroposterior roentgenogram of a first-stage dislocation. This characteristic change in outline from a demilune to a cone enables one to diagnose the lesion even from the anteroposterior *x*-ray plate. This diagnostic point has not been previously noted in the literature.

The *x*-ray appearance will, therefore, differ somewhat in the three stages. It is the first stage which is most readily overlooked. The anterior picture makes the diagnosis if one only knows what to look for. It was my associate, Dr. Roger T. Vaughan, who first called my attention to the characteristic change in outline which the bone undergoes during dislocation and which, so far as I know, has not been previously noted in the literature. (Compare Figs. 99 and 105.) Even in the lateral picture, because of the overlapping of the carpal bones, the displacement of the semilunar anteriorly may be overlooked by the surgeon who is not familiar with this lesion, or who is not looking especially for it. Even the novice, however, will make the diagnosis if, instead of the ordinary two-way plates, he has stereoplates taken in both directions—anteroposteriorly and laterally. Then the dislocation becomes as definite to his eyes, if the plates be well made, as if he had the dissected skeleton of the wrist set before him.

Normally, the distal concave surface of the semilunar fits into the proximal convex surface of the os magnum. When these two surfaces on the lateral x-ray plate can be seen to be no longer in contact, a dislocation may and must be diagnosed.

Even the second stage of this luxation is frequently overlooked. Here the separation of the semilunar from the os magnum is distinctly greater than in the first stage of the lesion, and the concave surface of the scaphoid, instead of being directed distally, is turned more or less anteriorly through an angle of 90 degrees or so. Even this amount of luxation, however, may produce no considerable anterior deformity, and may be overlooked by the surgeon not thoroughly familiar with the normal appearance of the carpal picture. But even the anteroposterior picture alone, because of the changed outline of the bone silhouette, leads the experienced surgeon to suspect the lesion and complete the diagnosis by making a lateral plate.

The third stage of the deformity, however, is readily recognized by any fairly competent man, often even without an x-ray plate. The marked anterior deformity just at or proximal to the wrist interval, the shortening of the carpus, the absence of the semilunar from its usual location, and the limitation of movement at the wrist-joint make a fairly typical picture.

The luxations of the hand have been known since antiquity, but they have not been recognized with frequency and accuracy until the Roentgen era arrived. Previous to the use of the x-ray, luxations of the hand were considered to be among the rarest of traumatic lesions. Dupuytren threw the great weight of his authority back of the statement that luxations of the wrist never occur—that the so-called luxations are always unrecognized fractures of the radius. That teaching was long accepted, but it has been utterly overthrown by the accumulated evidence of the x-ray examination. Even before the end of the nineteenth century Dupuytren's thesis was actively combatted by a few surgeons who had proved by autopsy the occurrence of wrist luxations; but even these surgeons considered the lesions extremely rare. Now the x-ray plate has demonstrated that the backward dislocations of the hand on the wrist, of which "semilunar dislocation" is the most frequent variety, are quite common lesions, making up 3 or 4 per cent. of wrist injuries.

Semilunar dislocation is the second most frequent injury of the carpus; but even so it is noticeably less frequent than fracture of the scaphoid. In something less than half the cases it is associated with fracture of the scaphoid at the latter's surgical neck.

What causes semilunar dislocation? The character of the trauma responsible for the injury is very similar to that which causes a Colles' fracture or a fracture of the scaphoid. Apparently it is not so much the nature of the trauma which determines the details of the injury as it is the anatomic structure of the individual wrist. The lesion, fracture or dislocation, whichever it may be, follows the line of least resistance in the wrist. When the bone is the weaker, fracture occurs; when the ligaments give way, there is a luxation. The accidents which most frequently produce the lesion are a fall forward on the hand, a sharp blow across the back of the wrist, or a cranking injury—back-firing of the motor on the down stroke of the crank. Of the cranking injuries, I have not seen so many instances since the electric starter attained its present vogue. The injury is most frequent in men, since they are the more exposed to physical violence, and in the laboring classes. It is usually seen in patients between twenty and forty years of age. I have never seen a case in a child or in a patient with epiphyses still ununited. Children who have a severe fall forward on the hands usually break the bones of the forearm well above the wrist or near the middle of the shaft.

Each hand is involved with about equal frequency in semilunar dislocation, and there have been a number of cases reported where both hands were involved simultaneously, a forcible argument in favor of an anatomic peculiarity being an important factor in the production of the lesion.

For diagnostic purposes it is best to consider these cases in two groups: First, the simple luxation, and, second, the luxation with extensive displacement of the semilunar. In making the diagnosis the history is not of much help, save as it calls our attention to the approximate location of the lesion. The same violence may produce a semilunar dislocation which produces a Colles' fracture, a scaphoid fracture, or a fracture of the semilunar itself. The patient comes to the surgeon complaining, as a rule, of deformity or nervous disturbances, due to pressure on the median nerve, or limitation of motion in the wrist. He does not frequently show the complete loss of use of the hand which is so common in Colles' fracture.

In carrying out the examination of the injured wrist it is advisable, first, to eliminate the other more common lesions, namely, fracture of the lower end of the radius and fracture of the scaphoid. This is done by eliciting certain negative signs: the normal position of the radial styloid, absence of tenderness over the lower end of the radius, and the presence of deformity *distal*, rather than proximal, to the bistyloid line, as in the case of Colles' fracture. However, a greatly dislocated semilunar may be drawn up into the wrist and lie, as in our case of combined semilunar dislocation and scaphoid fracture, proximal entirely to the bistyloid line. Again, tenderness over the ulnar styloid and signs of fracture do not rule out a semilunar luxation, because the latter lesion is usually accompanied by fracture of the tip of the ulnar styloid. Rupture of the internal lateral ligament is distinctly less frequent than the styloid fracture.

Some of the positive symptoms of this luxation we have already mentioned, but not in detail. The *shortening of the carpus* is best measured between the distal end of the radius and the proximal end of the head of the third metacarpal, doing the measuring on the dorsum of the hand, and comparing the two hands.

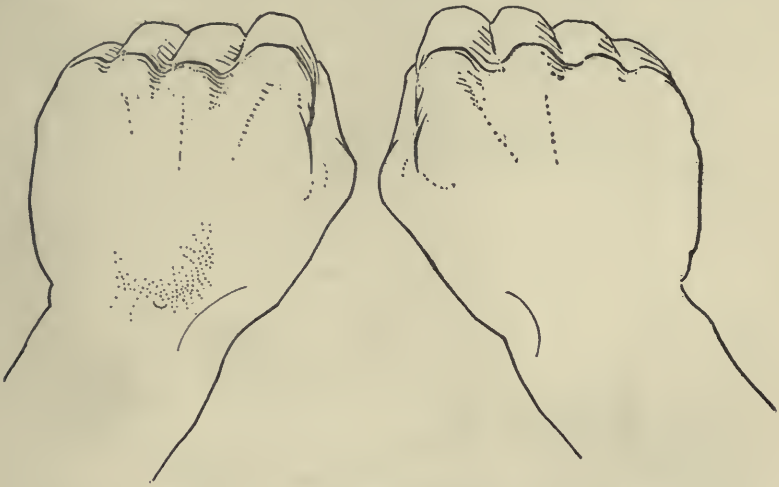
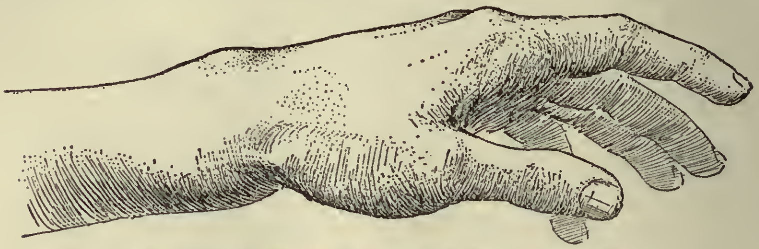


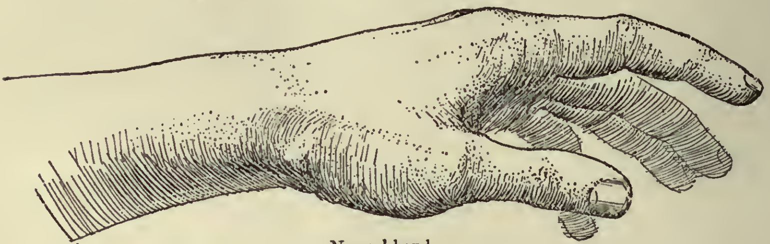
Fig. 106.—Showing the characteristic change in the knuckle outline due to dislocation of the semilunar and its consequent midcarpal shortening of the wrist. Normally, the middle knuckle projects beyond the others, but with dislocation of the semilunar, the middle knuckle (head of the third metacarpal) comes back to a level with, or even proximal to, the two adjacent knuckles.

The shortening is usually slight when the semilunar remains in place, that is, in the first stage of the luxation; but it may sometimes amount to as much as a centimeter or more when the semilunar is completely luxated, as in our third-stage case. This shortening of the carpus may be demonstrated without measurement by closing the fist and looking at the outlines of the knuckles. The third metacarpal bone will be seen to lie on a level with or even proximal to a line connecting the distal ends of the second and fourth metacarpals (see Fig. 106), an anomaly readily recognized by comparison of the two hands. Normally, the third metacarpal projects well beyond the second and fourth.

The visible *deformity*, while characteristic when typically present, is usually of not much aid in the diagnosis, save in the old cases. In the fresh cases the swelling present masks the topographic details. When, in the course of a month's time or so, the inflammatory reaction has disappeared, the patient may show either of two types of deformity, depending on the stage in which the luxation exists. In the first stage the deformity somewhat resembles "the silver-fork deformity" of fracture of the radius, save that the wrist is not quite so shortened as in a Colles' fracture, and that the dorsal hump lies distal to the wrist articula-



Forward luxation of semilunar bone with backward luxation of hand



Normal hand

Fig. 107.—Dislocation of the semilunar bone. The deformity is something like the "silver-fork deformity" of Colles' fracture, and is, unfortunately, very frequently mistaken for the latter. The dorsal prominence is made by the lower end of the os magnum turned backward. The palmar prominence, located between and obliterating the two palmar folds of the wrist, is due to the semilunar bone rotating forward and upward (*i. e.*, anteriorly and proximally). The dorsal prominence, however, lies distal to the line of the wrist-joint, while the dorsal prominence in Colles' fracture lies proximal to the joint, an important differential point in diagnosis.

tion. In the second and third stages of the deformity, however, when the bone has rotated well forward, it comes to lie as a projecting mass on the palmar surface of the wrist, usually between the two wrist-folds. Even this abnormal prominence may rather easily be overlooked unless careful inspection and palpation are carried out; but comparison with the other hand, if only the lesion is suspected, reveals the suspicious fulness just anterior or proximal to the normal location of the semilunar. In our most marked case the bony mass lay distinctly above the fold of the wrist-joint, and could be moved about like a bony tumor of the soft parts.

Pain is not a great feature in these cases, save in the few days following the injury and on the resumption of work. The old-standing cases of luxation are less painful, as a rule, than old fractures of the carpus, such as the ununited fractures of the scaphoid, for instance.

Along with the shortening of the carpus goes a thickening of it in its anteroposterior diameter, which may be both seen and felt, and measured, but may be overlooked on superficial examination, and is overlooked altogether too frequently, to the serious disadvantage of the patient.

The hand tends to lie in a position of semi-dorsal flexion, with half-flexed fingers—the so-called claw position. Volar flexion, both active and passive, is greatly hindered or impossible, and dorsal flexion is diminished and painful. On the other hand, the lateral movements of the wrist are usually free, and so are, of course, pronation and supination. The principal cause for the development of this variety of claw hand lies in the shortening of the flexor tendons by their becoming pushed forward by the anteriorly rotating semilunar bone.

Not only is the action of the tendons interfered with by the anterior dislocation of the bone, but the median nerve is frequently compressed and adds to the motor difficulties. The ulnar nerve, on the other hand, is very rarely involved, as one would expect from the anatomy of the region. When the median nerve is pressed on, sensibility is diminished in the territory of its distribution. The patient complains of cold, clammy fingers, which are cyanotic on examination. There are frequently also various paresthesias, particularly formication. Trophic changes occasionally develop. The skin becomes thin and shiny, and the nails striped and brittle. Some atrophy of the muscles may occur in long-standing severe cases, especially of the thenar eminence, when the median nerve is involved. The further advanced the deformity, the more marked the median nerve involvement is likely to be. It was a very annoying feature in one of our advanced cases.

In the rare cases where the ulnar nerve is involved there may be atrophy of the hypothenar eminence and of the interossei, an occurrence which adds still more to the claw-like appearance of the hand. In very old-standing cases the bones show an atrophy of disuse, as in similar chronic disabling lesions elsewhere in the extremities, particularly in patients of advanced years.

The *x*-ray plate, of course, is our ultimate recourse. It is the *x*-ray which has enabled us to make these diagnoses even in clinically unsuspected cases, and which has, on the other hand, taught us the clinical characteristics of the lesion and so enabled us often to make the diagnosis clinically in suspected cases, even without the picture's help. A two-way picture is not only desirable, but absolutely necessary, to make a diagnosis with absolute certainty, in some cases, and to exclude other wrist lesions. Of the two pictures, the lateral is the more valuable and decisive, whereas in Colles' fracture, for which this luxation is often mistaken, the anterior picture furnishes the more desirable evidence, and usually makes the diagnosis positively. Fracture of the scaphoid is also most easily diagnosed from the anteroposterior plate. It will be seen, therefore, how necessary it is to take two-way pictures, or, even better, two-way stereograms, of these injured wrists. Two-way pictures or stereograms are just as necessary here for accurate diagnosis as in the lesions of other complicated joints, of which the tarsus is an example.

Normally, the convexity of the os magnum fits into the concavity of the semilunar. When it does not, a luxation exists. This is the characteristic finding which the lateral *x*-ray plate gives us.

Now, what shall we do for these semilunar dislocations when we have diagnosed them? The treatment may be readily carried out, as a rule, if only the diagnosis is made. The diagnosis is the essential. Once that is made, the necessary line of treatment suggests itself naturally. One will use conservative measures first, particularly in fresh cases, and operative measures only as a last resort.

How does one reduce such a dislocation? The manipulation is not unlike that in the reduction of a Colles' fracture, partly manipulation and partly extension. In the first grade of the deformity the hand and wrist are grasped by the operator's two hands, just as in the reduction of a Colles' fracture. The patient's hand is extended and dorsoflexed; that is, the position produced by the trauma is reproduced. Then, with the fingers of the left hand, assisted occasionally by the hand of an associate, the semilunar is turned backward to its old position; and then, with vigorous extension, the hand is again brought forward, carrying the convexity of the os magnum into the concavity of the semilunar. In fresh or relatively fresh cases this manœuver will usually bring success, particularly if repeated a time or two. Occasionally, tissue or torn capsule may so intervene, particularly in those cases with much dislocation forward, that closed reduction becomes impossible. Then we must have recourse to one of two operative methods. We may either cut down upon the bone, remove the obstruction, and return the bone to its original position, or if, particularly in old-standing cases, this is impossible, through closure of the old semilunar cavity with scar-tissue formation, or the like, the bone may be excised *in toto*. It is surprising how little disturbance follows the excision of the navicular, and how rapidly a joint previously almost immobilized by the dislocation of the flexor tendons and pressure on the median nerve assumes active, vigorous, and well-nigh normal function.

With careful attention to the details of closed reposition and to the technic of manipulation, it is surprising how seldom one needs to excise these bones. It may be true, as some of the Germans insist, that the patients do as well without their semilunars and scaphoids as with them, and that primary excision is justifiable in both cases. In American surgery, however, conservatism is the policy. Our patients prefer to keep their anatomic belongings, and I believe it better surgery to spend time and trouble replacing the displaced bones where they belong rather than snipping them out as being of little consequence. Because the functional adaptability of the human body to anatomic changes is so excellent is no good reason for putting it to the test oftener than is absolutely necessary.

FRACTURE OF THE SEMILUNAR BONE

Fracture of the semilunar is one of the rarer lesions of the carpal bones, yet one occasionally meets it when studying all carpal injuries systematically with the *x*-ray plate. After having seen two or three of these cases, one can even suspect the existence of the lesion before the roentgenogram is made, particularly if the patient at the time of the injury was composed enough to note and remember the exact manner in which he fell.

The injuries which cause semilunar fracture are of about the same nature as those which cause a Colles' fracture or the other lesions of the carpus which we have just mentioned, namely, falls on the hand, cranking injuries, and the like. The fracture of the semilunar, however, has this one etiologic peculiarity: it occurs only in ulnar flexion, so far as I am familiar with the lesion. The reason for this fact is that in ulnar flexion the semilunar comes to lie between the concavity of the radius and the distal row of carpal bones, and, therefore, violence applied to the palm in this position of the hand finds the semilunar acting as a buffer between the radius and the rest of the carpus. In radial flexion of the hand, as you may remember, the scaphoid comes to lie in the concavity of the radius, and acts as the carpal buffer; but in ulnar flexion the semilunar lies wedged in between the concavity of the radius, on one side, and the convexity of the os magnum, on the other; and, held in position by the concave radial end, it is smashed by the impinging wedge of the os magnum.

Occasionally the injuries which cause semilunar fracture may be relatively slight as compared with the injuries causing dislocation of the same bone or fracture of the scaphoid. The trauma causing the two latter lesions is nearly always of a very severe nature, whereas occasionally a trifling blow or a slight fall from which one would expect only a sprain may result in a crushing or a linear fracture of the semilunar.

The clinical symptoms of semilunar fracture are more characteristic when a crushing injury is present—and this is by far the most frequent form of trauma—than when the fracture is simply transverse. With a crushing fracture there is usually a noticeable shortening of the carpus. This shortening may be actually measured by a tape, using the lower end of the radius and the head of the metacarpal as the points of mensuration, or one may note the proximal displacement of the head of the third metacarpal. Normally, the third metacarpal projects somewhat ahead of the second and fourth metacarpals when the fist is closed; but in semilunar fracture, just as in semilunar dislocation, the head of the third metacarpal comes to lie on a level with the two other bones or even proximal to them. Along with this shortening of the carpus goes a slight increase in the anteroposterior diameter; but this increase is by no means so pronounced as in dislocation of the semilunar. It is usually less even than we see in the first stage of semilunar dislocation. There is also less limitation of motion, as a rule, than with semilunar dislocation, and the limitation of dorsal is greater than of volar flexion. That physical disability is less in fracture as compared with dislocation is due to the absence of involvement of the flexor tendons in a fracture. Furthermore, the median nerve, too, is not involved with fracture, as a rule, and so one practically never sees the severe trophic disturbances in semilunar fracture which one occasionally sees with dislocation of the bone. Probably, however, the most important differential point between the dislocation and the fracture is the "tapping" test. This test is very similar to the "tapping" test which we spoke of in fracture of the scaphoid. You will remember that in fracture of the scaphoid, with the hand in radial flexion, there was intense pain produced by tapping on the head of either the second metacarpal or the third metacarpal, but the tapping over the heads of the fourth and fifth metacarpals was painless. In fracture of the semilunar in *ulnar* flexion intense pain is produced by tapping the head of the third metacarpal, but tapping on the head of the second metacarpal causes but little pain, and tapping on the head of the fourth perhaps just a little more than on the second. This tapping test is practically negative, usually, in dislocation; and the position of the hand in which the tapping on the third metacarpal is most painful serves to differentiate semilunar fracture from fracture of the scaphoid to a fair degree of certainty.

The *x*-ray plate, of course, is our final reliance. It is only by making it that we can be absolutely certain of the exact lesion present; and, remember, there must be a plate taken from each direction—one anteroposteriorly, and a second laterally. The anteroposterior plate shows not only the fracture, but also the variety of fracture—whether or not it is impacted. The lateral plate serves chiefly to exclude the possibility of semilunar dislocation.

All these niceties of differentiation are not purely a diagnostic exercise. They have intense practical significance. Unless these carpal injuries are accurately diagnosed, the proper treatment will not be instituted, and the patient will not secure a useful wrist. Unless treatment is instituted early in the case of a fractured semilunar, the result will be some disability of the wrist. Extension and immobilization is what should be practised. This is secured by attaching extension to the third finger, and at the same time immobilizing it. If the patient is allowed to go untreated, diagnosed as a sprain, for instance, the loose fragments of the crushed bone will play havoc with the wrist-joint. Then, when the patient has developed a severe traumatic arthritis, he returns again to the surgeon, who by that time can do nothing but excise the bone. Excision will remove the cause of the trouble and prevent its further development, but it will have little, if any, influence on the arthritic changes which have already developed. Such a patient will never again have a perfectly normal wrist, although it is possible to restore to it, with proper measures, a very considerable degree of usefulness.

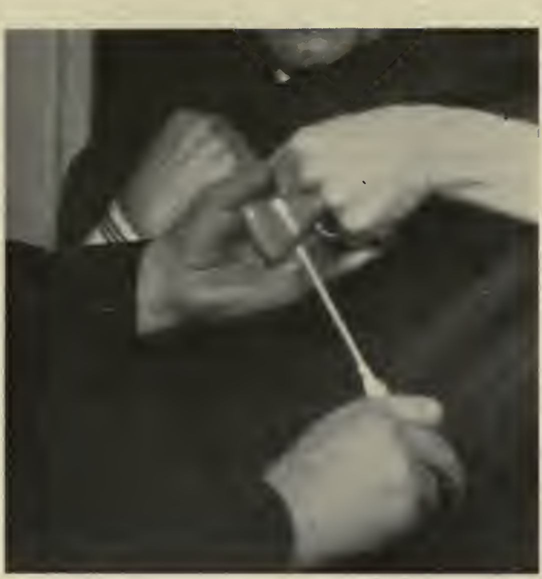


Fig. 108.—In fracture of the scaphoid, with the hand in ulnar flexion, percussion over the third knuckle produces no pain; but in semilunar fracture it produces more pain than percussion over the first knuckle does, although not quite so much as does percussion over the knuckle of the middle finger, because in the last instance the force of the blow is transmitted directly to the os magnum and so to the concavity of the semilunar.

It is astounding what errors both of diagnosis and treatment inexperienced surgeons may make in this region. Would you believe that a surgeon who had graduated from a reputable medical school could excise a normal semilunar bone when a fracture of the scaphoid was present and had been diagnosed for the surgeon by a competent röntgenologist? And yet that operator was a graduate of one of the best foreign medical schools. Simply he had been so long in general practice that he lost his bearings on opening the carpus,—an unfamiliar region to him,—and took out the wrong bone. Imagine his dilemma when the *x*-ray plate, taken after operation, showed his error!

A freshly fractured scaphoid without displacement should, I think, always be left in place. Union does sometimes occur, but if it does not occur, the pseudarthrosis which develops is often painless and the patient may have, so far as he knows, a perfectly normal hand. On the other hand, if pain does persist with the pseudarthrosis, and loose fragments of the bone appear to be present in the joint and are threatening to set up a chronic arthritis of the wrist, if one excises the scaphoid promptly, one has accomplished as much as by the early operation.

FRACTURE OF THE LEFT CARPAL SCAPHOID AND DISLOCATION OF THE SEMILUNAR

HISTORY

THE patient, a young woman aged twenty-two years, entered the hospital April 10, 1915. Her present trouble dates back to January 26, 1915, nearly three months ago. She was standing on a window-sill, washing the window from the outside. Some of the water which she was using fell on the sill unnoticed and turned to ice. She stepped on this spot, slipped, and fell to the cement walk, 10 feet below. The palm of the left hand and the left foot bore the brunt of the fall. She thinks she landed with her muscles rather relaxed, but is not very certain on this point, although she was not rendered unconscious. There was immediate pain and disability in the left wrist. The doctor who was called took her to the hospital, where an x-ray picture was taken within twelve hours of the accident. The plate showed a fracture and dislocation of one of the carpal bones—the patient doesn't know which one. The hand was manipulated a little and a palmar splint was applied. At the end of two weeks, because of the continued pain and disability in the wrist and the inability of the doctor to reduce the deformity, "even with a splint," a surgeon was consulted. He had some more x-ray plates made, diagnosed a fracture-dislocation of the scaphoid, and advised operation. The patient was accordingly operated on February 11, 1915, a little more than two weeks after the injury. The surgeon reported to us that the scaphoid was found fractured transversely and both fragments were dislocated forward. One fragment was reduced and the other was removed altogether. "It was hoped that the reduced fragment would remain in position and add materially to the strength of the hand." The wound healed by primary union, and the patient left the hospital February 19, 1915, with the hand and wrist in palmar and dorsal splints to "keep the bone in place."

Since that time, however, the pain has remained more or less constantly present, and the disability and deformity have persisted. The patient now comes to the hospital seeking relief for her still painful and utterly useless wrist.

COMMENTS

Dr. Murphy (April 12, 1915): This patient comes to us now, asking us to give her a good wrist, and yet she will not allow us to adopt the only means which can possibly be of any service to her—a cutting operation. Why? Because she has already been subjected to one unsuccessful operation, from which she received no benefit, and she is, therefore, thoroughly convinced that surgery can do her no further good. She brought her previous x-ray pictures with her, and we have had some more plates taken here—so we are in a position to give you all the

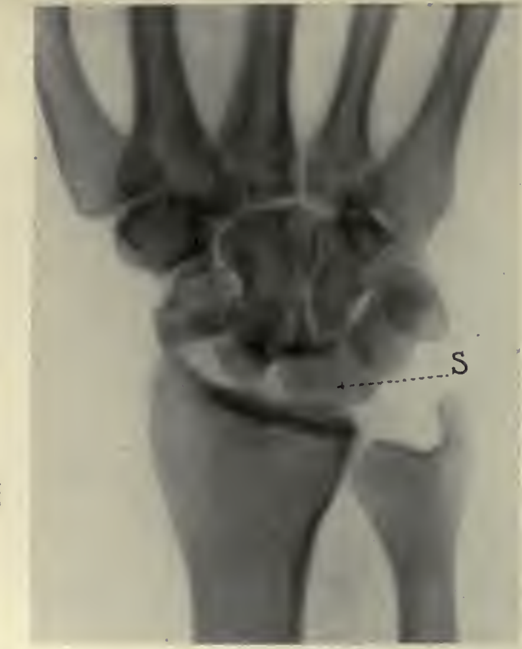


Fig. 109.—Transverse fracture of the carpal scaphoid and dislocation of the semilunar. Note the characteristic conic outline of the dislocated semilunar (S). The two fragments of the fractured scaphoid are widely separated, the proximal fragment following the dislocated semilunar, to which it is attached by the interosseous ligament. In this case the line of dislocation of the wrist has passed through the interval lateral ligament of the wrist, instead of through the tip of the ulnar styloid, as it did in one of our other cases and as is the rule.

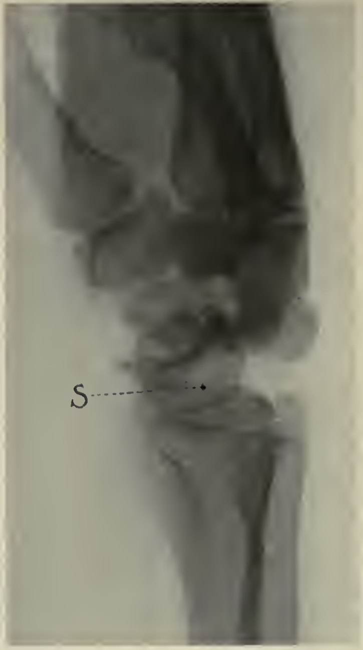


Fig. 110.—Transverse fracture of the carpal scaphoid and dislocation of the semilunar. Note how clearly the crescent-shaped outline of the dislocated semilunar stands out in this lateral roentgenogram (S). Note the convex proximal end of the os magnum lying just behind the posterior horn of the dislocated semilunar, whereas it fits normally into the semilunar concavity. A forward rotation of the semilunar has already occurred to the extent of about 30 degrees, *i. e.*, about half way between the first and second "stages" of the dislocation.

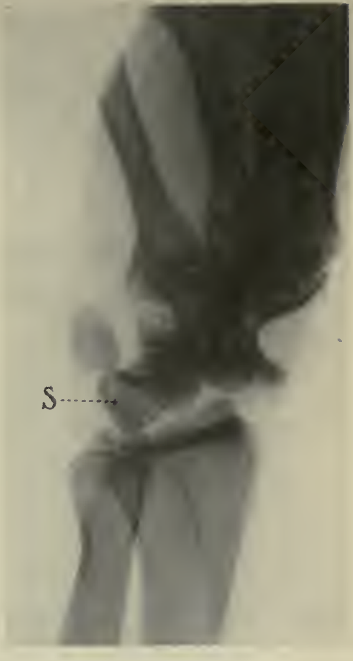


Fig. 111.—Transverse fracture of the carpal scaphoid and dislocation of the semilunar. This lateral roentgenogram was taken at a slightly quartering angle, and so the characteristic crescent of the semilunar is not seen. Nevertheless it can be made out lying in front of the os magnum if one will compare this picture with the other lateral view. The two fragments of the fractured scaphoid can also be made out.

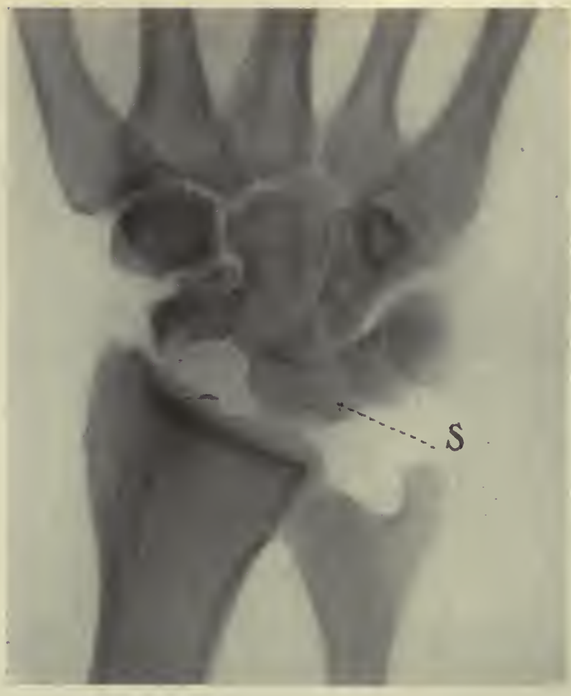


Fig. 112.—Transverse fracture of the carpal scaphoid and dislocation of the semilunar. Lateral roentgenogram after operation. Note the defect in the proximal row of carpals, due to the excision of the proximal fragment of the scaphoid. Note the characteristic conic outline of the displaced semilunar.

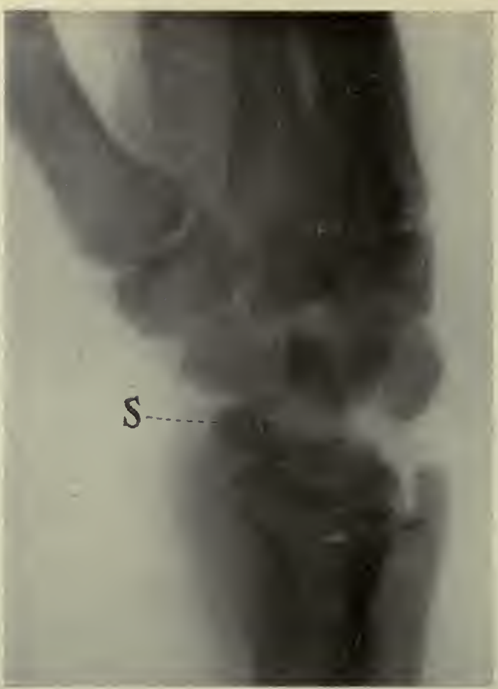


Fig. 113.—Transverse fracture of the carpal scaphoid and dislocation of the semilunar. Lateral roentgenogram after operation. Note that the dislocated semilunar (S) lies in practically the same position as before operation, except it is rotated a little more forward. The surgeon failed to reduce it because he did not recognize it.

facts in the case, and interpret just what happened. The first doctor who saw the patient tried to "reduce the deformity with a splint." *Never try to reduce a fracture deformity with a splint!* Nothing but disaster lies over that road! If you can't do it without, you can't do it with, a splint. That is the best way to produce pressure paralysis and Volkmann's contracture. You will never secure a reduction by that method, but you will often succeed in producing an ischemia of the part, with subsequent muscle and tendon necrosis. The doctor said there was a fracture of one of the carpal bones. His diagnosis was correct. There was; but there was more—there was an accompanying dislocation of the semilunar bone, which was entirely overlooked by the practitioner and the surgeon. Why did the surgeon fail to recognize the condition present? Because he did not know better? No, I think not, because I personally know that he has had experience in just this line of work. He simply was not cerebrating when he examined those *x*-ray plates, because they tell the story, and they tell it without the shadow of a doubt. When the surgeon operated, what he really found was not two fragments of a fractured and dislocated scaphoid, but the proximal fragment of a fractured scaphoid attached to the dislocated semilunar. But once again he was on a mental vacation. He excised the dislocated scaphoid fragment and attempted to replace the dislocated semilunar, but mistaking it for the other fragment of the scaphoid, he tried to manipulate it into the scaphoid socket, where it couldn't possibly fit, instead of trying to bring its concave upper surface in contact with the convex lower surface of the os magnum, as might perhaps have been done at that time had the condition been thoroughly understood. The reduction not having been made, the splint and dressing subsequently used were of no value, and the patient comes to us now with a still incapacitated wrist, but so "surgery shy" that she balks at the only possible means which can give her relief.

What should be done for her now? The displaced semilunar must be excised. At this late date it will not be possible to replace it in its normal relation because of the scar tissue which has formed there. What is the outlook in this case? It would be good if she would only allow us to do what is necessary for her. It is surprising how well most of these patients get along without a semilunar. So long as the displaced bone remains pressing against the median nerve and the flexor tendons, pain will persist, and both flexion and extension will remain greatly limited. The patient will not use the affected hand, and the longer it remains unused, the stiffer and more useless it will become.

Note the history states that the patient fell on her left side, and broke her fall with her left hand and foot. If you will place yourself in this position, you will note that the left hand under such circumstances turns in radial flexion. That fact explains why the patient suffered a scaphoid fracture along with the semilunar dislocation, because it is chiefly when the hand is in radial flexion that fracture of the scaphoid occurs. When in this position it acts as a buffer between the lower surface of the radius, on the one hand, and the convexity of the os magnum, on the other.

What a valuable and indispensable aid to us the *x*-ray has become! How easy it is now, when one has had the necessary experience, to interpret cases like this from the *x*-ray plate, and how difficult it was, before the *x*-ray came into use, to analyze such a case properly. Only those surgeons who have worked under both conditions can really appreciate the great advance which the *x*-ray has made possible in the recognition and treatment of these conditions. This case is particularly interesting and instructive because similar injuries are so frequently overlooked and improperly treated by the general surgeon, as well as the family physician. The result of failure to diagnose the condition in time is disastrous. Every physician as well as every surgeon should be aware of the frequency of these lesions and know their clinical signs and the *x*-ray appearances just as intimately as he does the signs, symptoms, and *x*-ray findings of the more common Colles' fracture. If one is informed about these lesions and is on the alert for them, he cannot overlook them.

[NOTE.—The patient has not yet been able to make up her mind to have the necessary operation performed.—ED.]

DISLOCATION OF THE LEFT UNCIFORM BONE

HISTORY

The patient, a young man aged twenty-two years, entered the hospital December 1, 1910, with the following history: His present trouble dates back to a time, three months ago, when he caught his left hand between two rollers, severely and painfully crushing it, but not breaking the skin. Immediately following the injury movements of the hand or wrist caused him severe pain, and on the next day the hand and forearm were both considerably swollen. On the second day after the accident, because of the continuance of the swelling and disability, he consulted a physician, who told him that the small bones of the wrist had been broken. The doctor applied an anteroposterior splint and kept the wrist immobilized in it for two weeks. After the removal of the splint passive motion was instituted, and the patient then noticed for the first time a small, hard projection upon the dorsal surface of the wrist, toward its ulnar side, about one inch above the styloid process of the ulna, and a slightly lesser distance from the ulnar border of the hand. The swelling of the hand and forearm soon disappeared, but this bony projection did not, nor did the patient subsequently regain full use of his hand and wrist. Flexion and extension of the wrist cause him considerable pain, and are distinctly limited in range, especially flexion. Both abduction and adduction of the wrist are somewhat painful and limited in degree.

COMMENTS AND OPERATION

Dr. Murphy (December 3, 1910): This is a very unusual case, and for that reason of great surgical interest. So far as I have been able to look up the literature there are only three cases of this rare lesion on record to date. They are: (1) The case of Buchanan (*British Medical Journal*, 1869, page 398), where the lesion was part of a compound injury of the carpus; Oberst (*Fortschritte auf dem Gebiete der Roentgenstrahlen*, 1901, Erg. Heft, 5) and Eigenbrodt (*von Bruns' Beiträge*, vol. xxx).

Probably the violence of the injury and its peculiar action—the hand being caught between two rollers—were responsible for this particular lesion. It is the first of the kind that I have ever seen, but there was very little difficulty in diagnosing it, because, like so many of the rare lesions, it presented definite and characteristic features quite unlike any of the common lesions in this location. The bony prominence here is dorsal, whereas in most of the common carpal dislocations, semilunar and scaphoid, it is palmar. It is located on the ulnar side of the hand, whereas the other common dislocations occur in the middle or on the radial side of the hand. The bony projection is in line with the distal row of carpals, while the common dislocations involve the bones of the proximal row. Note, too, that when the patient closes both fists and holds them together for comparison the last knuckle of the left hand lies further proximal to the line of the other knuckles than does the last knuckle on the right hand; that is, there is a distinct shortening of the carpus on the ulnar side of the injured hand. So you see we had a very good idea of the nature of the lesion even before the *x*-ray plates were made, and yet we had never seen such a case before. The *x*-ray pictures clinched the diagnosis. We have had both plates put in the shadow-box, where you can see them now. In the postero-anterior picture note that, although the unciform lies approximately in its normal position, it distinctly overlaps the adjacent os magnum and the bases of the last two metacarpals. The lateral view is the more distinctive, because it shows the intact unciform lying almost entirely dorsal to the rest of the distal row of carpals, and its outline shows no break in continuity; that is, it is a dislocation uncomplicated by fracture. Note that all the other carpal bones are normal in contour and position, that is, there is no sign of any other accompanying fracture or dislocation of the carpal bones; the pisiform, cuneiform, and os magnum all appear normal.

What shall we do for this patient? There is only one thing which we *can* do for him, at this late date,—three months after the injury,—that is, excise the projecting bone. It has been causing painful tingling, and, lately, some numbness on the back

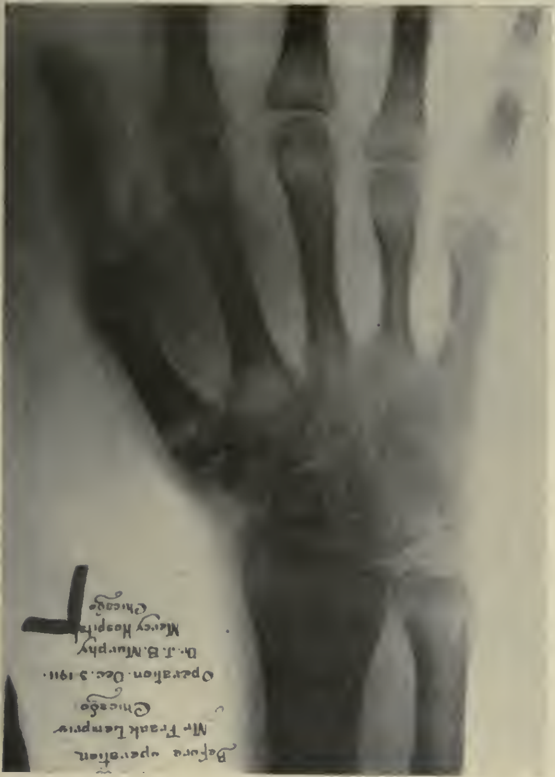


Fig. 114.—Dorsal dislocation of the unciform bone. The postero-anterior roentgenogram does not give so characteristic a picture of the deformity as the lateral roentgenogram; but, nevertheless, it shows the displaced unciform quite clearly overlapping the surrounding bones, os magnum, and the heads of the fourth and fifth metatarsus, considerably more than it should.

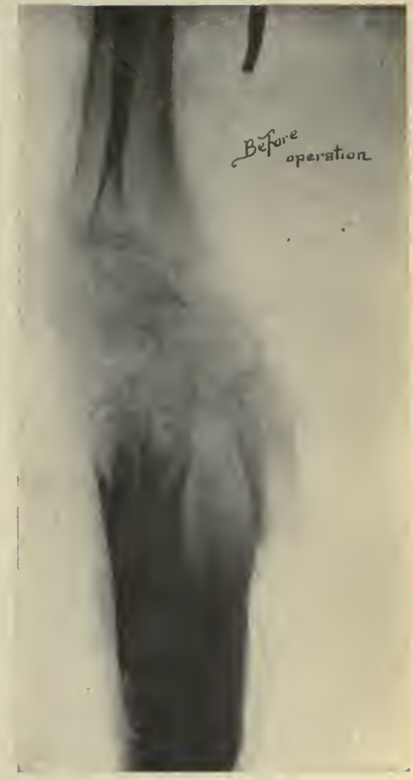


Fig. 115.—Dorsal dislocation of the unciform bone. Note the projection which it forms beneath the dorsal tendons, its characteristic outline, and the absence of any lesions of the other carpal bones. Lateral roentgenogram before operation.

of his last three fingers, probably because of pressure on the dorsal branch of the ulnar nerve, where the latter passes around to the back of the wrist. The bone has also pushed back the extensor tendons of the fourth and fifth fingers, and so prevents complete closure of the hand; and it also partially locks the joint in ulnar, radial, and dorsal flexion.

[The bone was excised through a dorsal longitudinal incision, the dorsal branch of the ulnar nerve and the dorsal tendon being carefully displaced, and injury to them avoided during the dissection. The wound was closed with horsehair, and the usual gauze dressing and a dorsal wood splint were applied.]

[Note.—The wound healed by primary union, and the patient left the hospital December 22, 1910. We have heard no complaint from him since then, and so presume that he secured a good result—as he should have—and felt well satisfied with it.—Ed.]

TRANSVERSE FRACTURE OF THE LEFT CARPAL SCAPHOID, SEMILUNAR DISLOCATION, AND FRACTURE OF THE ULNAR STYLOID

HISTORY

The patient, a man aged forty-two years, entered the hospital November 11, 1910, with the following history: On June 17, 1910, while he was 12 feet up on a ladder, it fell and the patient struck the ground heavily with the palm of the left hand, the arm being extended. He was not made unconscious and was able to rise promptly unassisted. He suffered a severe injury of the left wrist, however, including a laceration of the skin, on its anterior surface, about half an inch long. It caused him great pain and he was unable to move it. In about thirty minutes it became noticeably swollen; so he went to a hospital, where the wound was dressed, but nothing further done. The wound subsequently healed without suppuration. Since the accident the patient has continued to have pain and disability in the wrist, and the normal contour of the joint has never been regained.

EXAMINATION BY DR. MURPHY, NOVEMBER 11, 1910: The patient as he fell evidently turned partly on his left side, that is, with his injured hand in radial flexion, a position which predisposes to scaphoid fracture and also to semilunar dislocation, both of which evidently have occurred here, together with a tearing-off of the styloid process of the ulna. There is a bony projection on the anterior surface of the wrist—distal, not proximal, to the lower end of the radius, which is apparently intact, perfect in outline, and without any tender points. This bony projection presses upon the median nerve, causing pain in the hand, and has also pressed the flexor tendons forward, preventing extension of the wrist, and also interfering with flexion. Both ulnar and radial flexion, too, are somewhat limited and painful. The normal depression in the "anatomic snuff-box" is obliterated, and there is marked tenderness here over the broken scaphoid, more in radial than in ulnar flexion. Jarring of the first and second knuckles of the closed fist in an upward direction is quite painful. There is some soft, cartilaginous crepitus in the joint, but no characteristic bony crepitus. There is a noticeable shortening of the left carpus as compared with the right, and the strength of the left hand is greatly impaired. The loss of strength is particularly noticeable in the grip. The ulnar styloid can be felt as a separate, somewhat movable, and tender piece of bone, just beyond the lower end of the ulna.

The *x*-ray plates completely confirm the conclusions based on our physical examination. There is a transverse fracture through the body of the scaphoid and a dislocation of its proximal half, together with the semilunar bone, which is in the position known as the first stage of dislocation. The ulnar styloid is transversely fractured and separated by an interval from the head of the ulna. Since so long a period of time has already elapsed, any procedure other than open operation and excision of the fragments is out of the question, and we must, therefore, remove the proximal fragment of the scaphoid, together with the dislocated semilunar.

[NOTE.—The patient was operated Saturday, November 12,

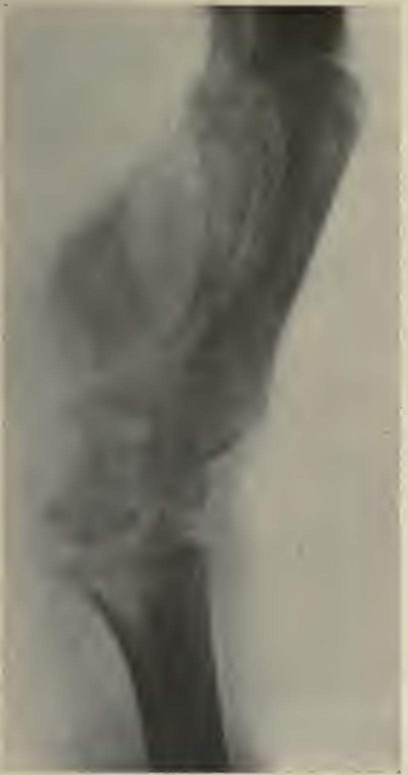


Fig. 116.—Transverse fractures of the scaphoid and tip of the ulnar styloid and dislocation of the semilunar. The patient moved slightly while this lateral roentgenogram was being made, and so obscured its details. The dislocated scaphoid can be seen, however, lying on the palmar surface of the wrist, with its concave surface still facing distally, but entirely out of contact with, the convex surface of the os magnum.

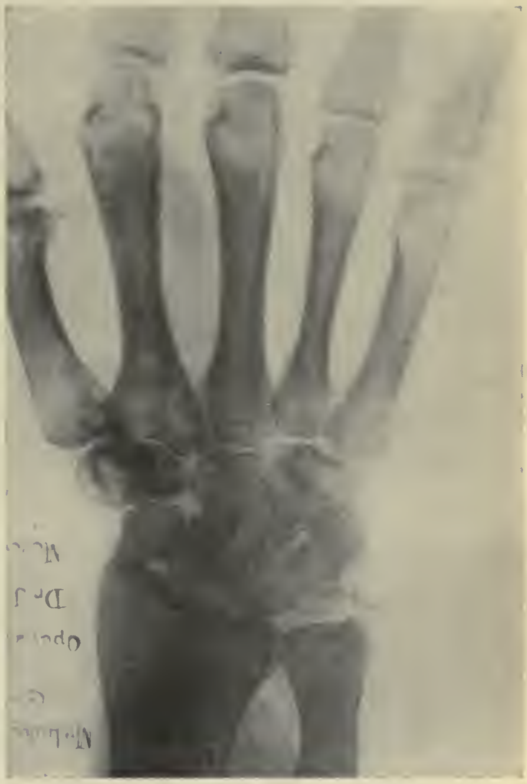


Fig. 117.—Transverse fracture of the scaphoid and tip of the ulnar styloid and dislocation of the semilunar bone. Postero-anterior roentgenogram before operation. Note the two fractures and the characteristic conic outline of the semilunar bone in the "first stage of dislocation."

1910. The proximal half of the scaphoid and the dislocated semilunar were removed, as we had previously planned, through the usual anterior longitudinal incision. The wound healed by primary union. The stitches were removed a week later, and the patient was permitted to go home on November 19, 1910.—Ed.]

DISLOCATION OF RIGHT SEMILUNAR BONE AND FRACTURE OF RIGHT ULNAR STYLOID TIP. —FRACTURE OF LEFT ULNA IN ITS UPPER THIRD

HISTORY

The patient, a young man aged twenty years, entered the hospital December 15, 1914. His family and personal histories are negative.

His present trouble dates back to July 25, 1914, when he was suddenly thrown from his motor-cycle. He thinks he struck on his shoulders, but he was too dazed by the accident to be certain. From the nature of the injury it is evident that he must have struck the ground with his hands or with his hands and left elbow. He was not rendered unconscious by the accident, but arose immediately and shut off the engine of his motor-cycle. He could use only his right hand to do this, because his left hand and arm had been disabled by the accident. He went to the nearest doctor, who applied a temporary splint to the left forearm, which he said was broken. He then went to his brother, who is a physician, and the latter took him to a hospital, and, with the assistance of another doctor, reduced the fractured elbow, manipulated both wrists, and dressed the elbow in flexion at an angle of about 90 degrees, retaining it in position with splints. The patient's brother said that there was a dislocation of both wrists and a simple fracture of the ulna in its upper third.

The splints were removed from the elbow at the end of nine days, and the forearm at the site of the fracture was seen to be swollen and greatly discolored. A plaster-of-Paris cast was then applied, with the elbow at a slightly obtuse angle, and the elbow was retained in the cast for four weeks. When the cast was removed motion in the elbow-joint was very limited—both flexion and extension; but since passive movement and massage have been instituted, the range of motion has been increased to about 15 degrees. Pronation and supination were and have remained practically absent. The left wrist-joint appeared to be normal in contour when the splints were removed, and its movements were free. (Therefore it probably was not dislocated, but only sprained by the accident.) There have been no disturbances of sensation or motion in the left hand. The fingers all move perfectly.

The right wrist was put up in a splint at the same time that the left elbow was dressed. The splint was removed on the second day because of the great pain which it caused. The wrist was then tightly bandaged, although greatly swollen and discolored at the time.

Examination by Dr. Murphy: There is an oblique fracture of the ulna, splitting off the upper end of the ulna, including the olecranon process. Some small fragments have been split from the outer side of the olecranon, and the coronoid process seems to be somewhat changed in contour, and seems to possess a small exostosis on its anterior lip. The head of the radius appears normal, except for some deforming arthritic changes. There is a slight luxation backward of the upper end of the ulna, including the olecranon process. Some bony union has taken place, but it is not yet complete.

There is also an anterior dislocation of the right semilunar bone. Its characteristic crescent shape shows in the lateral *x*-ray plate. The postero-anterior plate shows the characteristic conic-shaped shadow overlying the shadows of the unciform, cuneiform, and os magnum. The semilunar bone has rotated forward through an angle of not quite 90 degrees, and therefore this cannot be called a second degree dislocation, although it is almost one. The other carpal bones appear normal, and their interrelationships appear normal. The dislocated bone lies

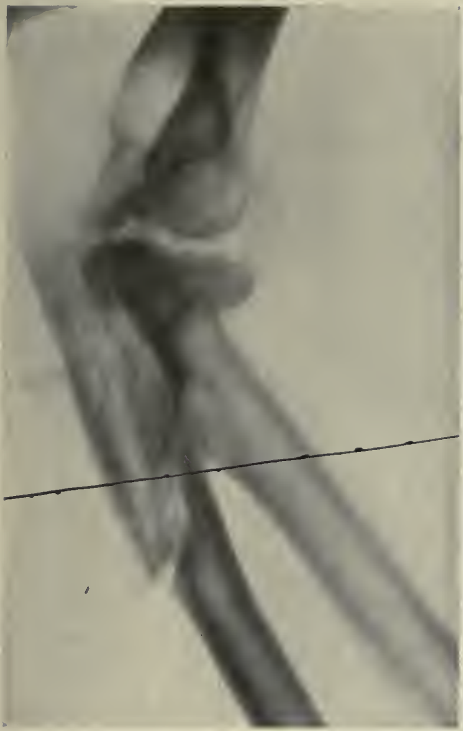


Fig. 118.—Oblique splitting fracture of upper third of left ulna. Exostosis formation on coronoid process. Lateral roentgenogram made nearly four months after the injury. (The plate is blurred, owing to movement of the patient during the exposure.)

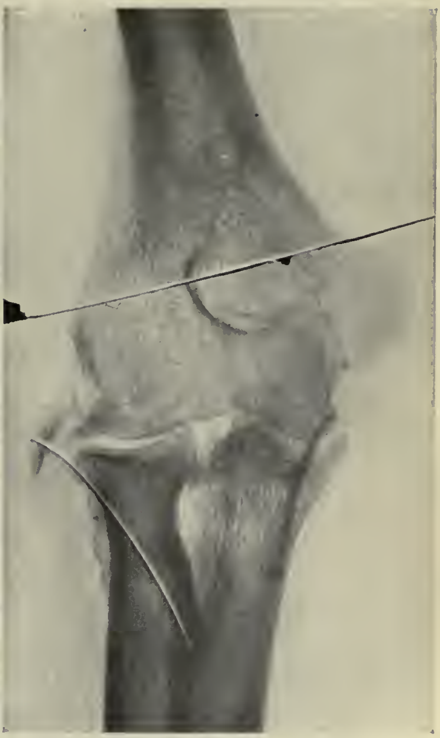


Fig. 119.—Oblique splitting and comminuted fracture of the upper third of left ulna. Anteroposterior roentgenogram made nearly four months after the injury.

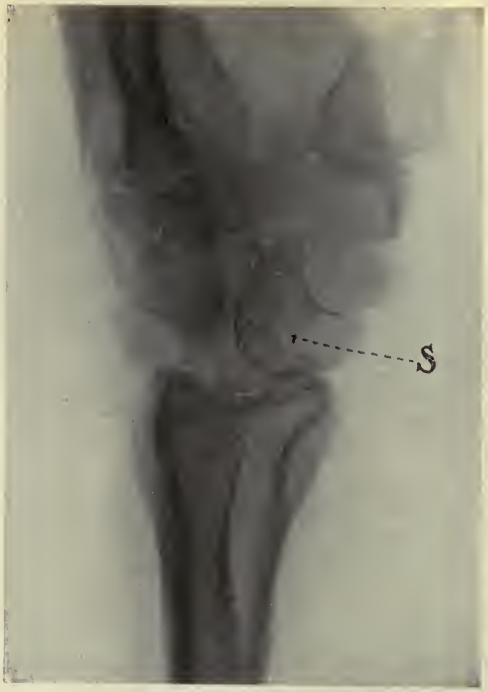


Fig. 120.—Dislocation of the semilunar bone and fracture of the tip of the ulnar styloid. Note the crescent-shaped outline of the dislocated semilunar, which is already rotated forward more than 60 degrees, but not quite 90 degrees. Therefore it is not yet a "second stage dislocation," but almost one. The details of this lateral roentgenogram are not quite sharp because the patient moved slightly while it was being made.

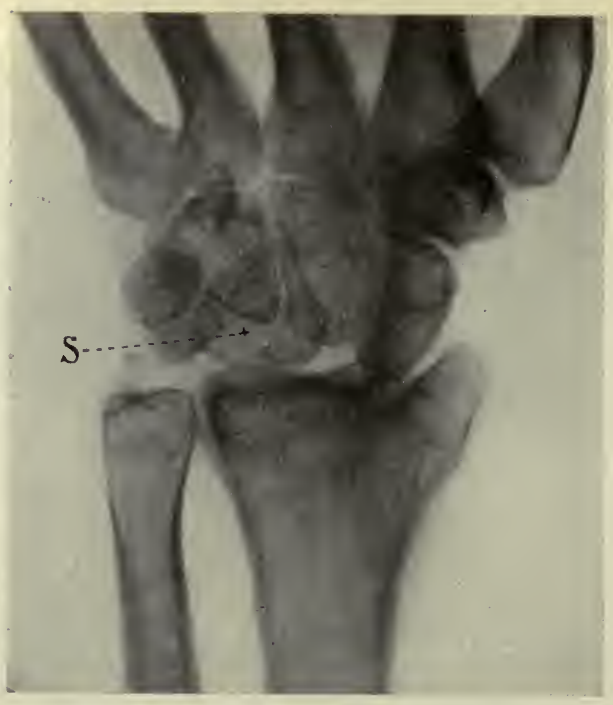


Fig. 121.—Dislocation of the semilunar bone and fracture of the tip of the ulnar styloid. Note the conic outline of the dislocated semilunar, characteristic of the first stage of the luxation. The diagnosis can be made positively from this postero-anterior radiogram alone, but should always be verified by having the lateral plate also made. For the proper study of every fracture and dislocation plates made from at least two different directions are essential.

beneath the flexor tendons and the median nerve, on both of which it exerts injurious pressure. The fingers cannot be fully extended or flexed, although they can be moved slowly in all directions. Flexion of the wrist is almost nil, only about 5 degrees remaining present. There is a numbness and partial anesthesia of the index- and middle fingers on the right hand, due to the pressure of the dislocated bone on the median nerve.

An open operation will probably eventually be necessary to remedy the limitation of motion at the elbow, but we shall not undertake it now. It will be better to wait until we have treated the wrist before removing the excess of callus at the elbow and the malunion which is interfering with movement there.

The anteriorly dislocated semilunar bone must be removed from beneath the flexor tendons, where it is interfering with their movement, as well as pressing on the median nerve.

COMMENTS AND OPERATION

Dr. Murphy (December 16, 1914): This patient may have a trifling splitting fracture of the head of the radius, as well as a fracture of the upper end of the ulna, with more or less comminution, but I am more inclined to believe that the radius is intact. The tip of the coronoid process is elongated somewhat anteriorly, like an exostosis. These partial ankyloses of the elbow are very difficult lesions to treat satisfactorily. After one has performed the open operation and has removed all the excessive callus, together with the underlying periosteum, one may be very proud of his work, but not for very long. In six, eight, ten, or twelve weeks the patient will usually return with an exostosis larger than the original. Then one will not feel nearly so satisfied. But the surgeon may then operate again, and this time the patient is very likely to return with a complete ankylosis of the joint because of the still more excessive formation of callus. I merely cite these innocent-looking traps for the unwary in order to warn those who have not already had one of these unpleasant experiences. Our only task to-day is to correct the deformity in this right wrist and restore its function.

Notice the characteristic deformity of this wrist. It looks very much like the "silver-fork" deformity of a Colles' fracture, and yet it is not difficult to differentiate it from the latter, because the resemblance is, after all, only superficial. Here the "silver-fork" projection on the back of the wrist is due to the lower end of the os magnum, which projects backward, and lies an inch or so more distal than does the backward projection in a Colles' fracture. Moreover, there is an anterior projection present here (due to the dislocated bone) which obliterates the two folds on the palmar surface of the wrist, and makes the wrist thicker than normal in its anteroposterior diameter, something which one does not see in a Colles' fracture. If you will look more closely at the back of this hand, you will note that the knuckle of the middle finger does not project beyond the line of the other knuckles, as it normally does, but lies parallel with them or a little behind. This phenomenon is due to the shortening of the carpus caused by the luxation forward of the semilunar bone and the occupying of its former site by the lower end of the os magnum, to which the middle metacarpal is attached. Furthermore, one notices, on carefully palpating the lower end of the radius, that the bony landmarks there are intact; and, if this patient were awake, this palpation of the radial head would cause no pain, as it always does in a Colles' fracture. In this particular case there was still a little tenderness over the styloid tip of the ulna, which was fractured at the time of the original accident, and has not yet reunited. The disturbance in the function of the median nerve, that is, the numbness in the middle and the index-finger of which this patient complains, is due to pressure on the nerve by the dislocated semilunar bone; but the same disturbance of sensation is occasionally seen with a Colles' fracture, and in a Colles' fracture the ulnar nerve is more frequently involved than it is in dislocation of the semilunar bone, where ulnar involvement is relatively rare.

Note the characteristic outline of the dislocated bone in this postero-anterior *x*-ray plate. Normally, in this frontal picture one sees the semilunar bone throwing a rather typical crescent-shaped shadow; but as soon as the semilunar becomes rotated somewhat forward, as well as dislocated, its shadow assumes this

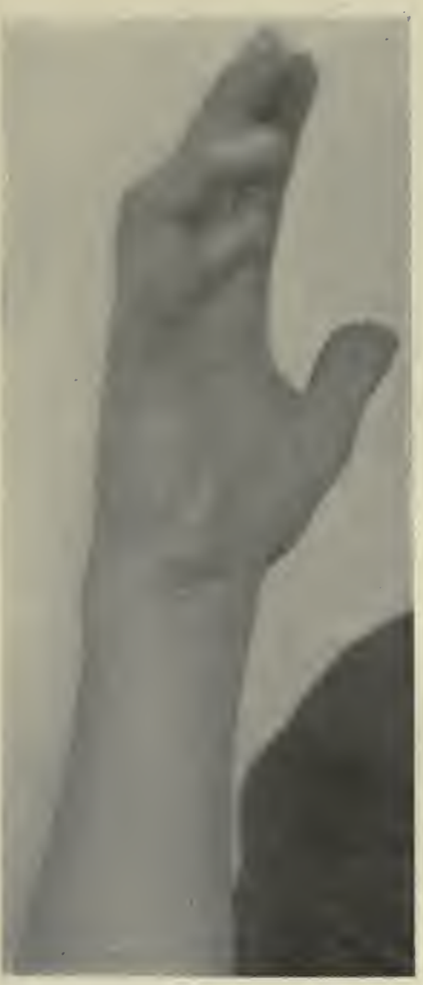


Fig. 122.—Dislocation of the right semilunar bone (nearly second stage). Note the palmar protuberance at and slightly distal to the line of the wrist-joint; the increase in the anteroposterior diameter of the wrist; and the stiff extended position of the hand and fingers. Photograph made on day before operation, nearly five months after the injury.

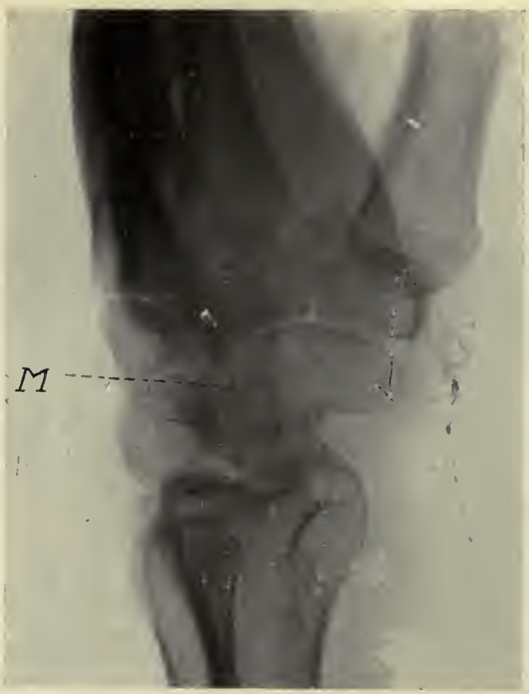


Fig. 123.—Dislocation of the semilunar bone and fracture of the tip of the ulnar styloid. Lateral roentgenogram after excision of the luxated bone.

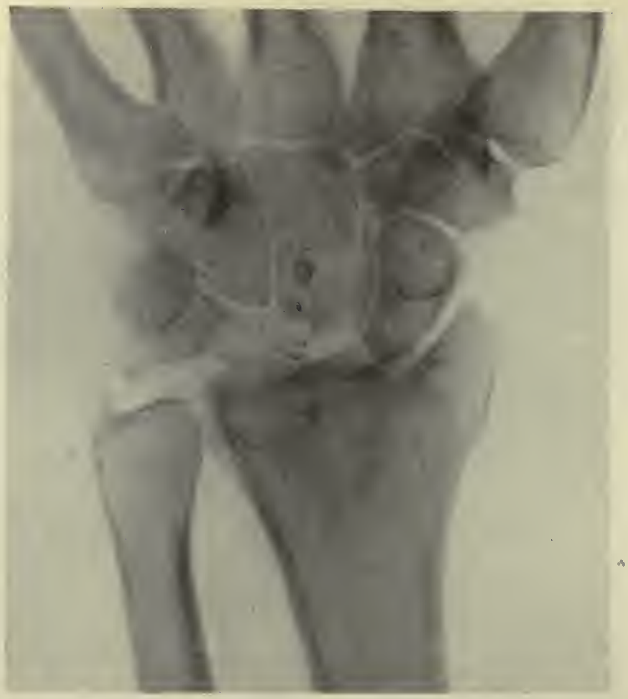


Fig. 124.—Dislocation of the semilunar bone and fracture of the tip of the ulnar styloid. Posteroanterior roentgenogram after excision of the luxated bone. Note that, except for the defect in the proximal row of carpals caused by the removal of the semilunar, the carpus is normal in appearance, for the remaining bones are held in their normal interrelationships by the strong intercarpal ligaments.

conic shape which one sees so beautifully in this x-ray plate. In such a typical case one can make the diagnosis from the postero-anterior roentgenogram alone, although in all wrist injuries, for the sake of ruling out other possible lesions, two-way plates must always be taken.

The lateral x-ray plate is even more characteristic than the postero-anterior plate. In it you notice the crescent of the semilunar tipped forward at an angle of nearly 90 degrees, instead of reposing, as it normally should, with its crescent directed distally and with the bone lying between the proximal end of the os magnum and the distal surface of the radius. This is almost what one would call the second stage of dislocation, which is usually defined as a 90-degree rotation forward. The first stage, you remember, is a simple dislocation, where the concave surface of the crescent is still directed distally. When the dislocated bone rotates still further forward, owing to the attachment of its anterior horn to the anterior surface of the lower end of the radius, the crescent of the bone comes to lie still further forward and upward, and the rotation may amount to as much as 180 degrees at times, as, for instance, in a case which we had recently. (See this (June) number of the CLINICS.)

A dislocation which has lasted as long as four months, such as this man has, becomes impossible to reduce, because the cavity which the bone formerly occupied is filled partly with connective tissue and partly by the encroachment on it of the adjacent bones. There is only one thing to be done now, and that is to remove the bone entirely, for it is acting only as a foreign body in the wrist, interfering with the movements of the flexor tendons, and pressing on the median nerve.

Transverse fracture of the scaphoid is a more common injury than dislocation of the semilunar. It is only a year ago that we had one of the students from the University of Wisconsin, at Madison, who suffered a transverse fracture of the scaphoid, together with a semilunar dislocation. The two fragments (the semilunar and the proximal fragment of the scaphoid) were luxated forward and upward into the wrist, attached to each other, and they both finally had to be removed in order to restore the function of the flexor tendons and the median nerve. As is usually the case with these injuries, the lesion was not recognized in his home town, and therefore the treatment instituted there was a complete failure. We had another similar case not long ago. The patient came from Dubuque, and he also had had previous attempts made to correct the deformity. Pulleys were used, and all the sorts of apparatus which an ingenious surgeon could think of to try to force back the projecting bony mass. Some of these apparatuses and pulley arrangements reminded me very much of the harnesses which horsemen use for restraining and training balky horses. Such apparatus traumatizes the tendons and nerves very severely, but does nothing in the way of accomplishing reduction.

[Dr. Murphy makes the usual longitudinal incision on the palmar side of the wrist over the apex of the bony mass.]

We are retracting the edges of the wound and now the projecting tendons come into view. I am slipping my finger up underneath the tendons into the space which the semilunar formerly occupied. Note that the tip of my finger will no longer fit into this space, which has been extensively encroached on by the cuneiform, os magnum, and scaphoid. It is impossible to replace the bone, no matter how much we might try to distend this space. Therefore we shall follow our contemplated plan of excising the bone. I am passing in my dissection to the ulnar side of the flexor tendons, and to the outer side of the flexor carpi ulnaris, and am working the semilunar bone loose by this route of approach. Now you can see it sticking up in the field just to the inner side of the flexor tendons. One can readily understand why the patient could not flex or extend his fingers. There was no power in the flexor tendons because they had to work over the edge of that bone, just as a rope works over the edge of a pole when a half-turn is put on it. You know how easily a child even can stop a colt or a horse from running by just giving the halter a half-turn around a hitching-post—not a half-hitch, but just a half-turn. That is what has happened here. Now that the semilunar is in view, note that its articular surface is turned forward into the palm at an angle of almost 90 degrees from its normal position, just as the x-ray indicated. It is three months since the accident, and therefore the bone is fairly firmly fixed in its new position by connective tissue. As I am dissecting it free, note how intimately its anterior horn is connected by this ligamentous band with the anterior lip of the lower end of the radius. I must chisel off the anterior surface of the tip of the radius in order to separate this ligament completely, and to make the surface of the radius perfectly smooth.

The semilunar is now completely excised, and we have only to remove the constrictor before closing the wound. We are closing the skin with an ectropion stitch, so that the subcutaneous fat will lie next to the palmar surface of the tendons in every spot; and therefore the tendons will not unite subsequently with the skin. We shall seal the wound with collodion, and dress the hand out straight, with the fingers extended. You see that the palmar protuberance now is entirely gone, and that the anterior surface of the wrist has regained its normal flat contour. The tendons have been in this contracted position for a long time, and therefore we must straighten the hand to the full extent, and also loosen up the contracted capsule of the wrist a little. Note that we carried out all our operative procedures without touching the wound with our gloved fingers or with any part of the instruments which had touched our fingers. If we obtain the result here which our efforts deserve, the patient should secure a wrist with almost perfect function.

[NOTE.—The wound healed by complete primary union. After the stitches were removed, gentle passive movements and massage were instituted, and when the patient left the hospital, about two weeks and a half after the operation, the movements in the wrist and fingers were already very satisfactory and very much better than before operation. He should obtain a practically perfect result.—ED.]

A TALK ON APPENDICITIS

Dr. Murphy (April 7, 1915): Just recently a critic took a Chicago surgeon to task in the columns of a medical journal because the latter had published a colored picture of a gangrenous appendix in connection with a practical talk on its proper treatment. The critic intimated that appendicitis was ancient history. It *is* ancient history; but does that statement mean that all practitioners are masters of the subject, or that the disease is efficiently handled at the present time?

In looking up recently for the Year-book of Surgery the hospital statistics on the results of operations for appendicitis, what mortality rate do you suppose I found—the hospital mortality rate? Some one guess! I do not mean the mortality for one, two, or three splendidly equipped hospitals, with staffs composed of the leaders of the surgical profession—I am speaking of the combined statistics of a number of hospitals in the United States. You know how perfect many of us think we are in this line of work, and how we fold our arms across our gowned but protruding chests after extracting an appendix and mentally, at least, exclaim, like little Jack Horner, "Oh! what a great man am I!" *That average hospital mortality rate is just a little over 10 per cent.!* If that is not a solar plexus blow to our conceit! These are not surgeons' statistics: they are the statistics of hospital managements, figures taken from the printed annual reports of hospitals which are progressive enough to publish reports. They include appendicitis cases of all classes brought to the hospital for operative treatment.

Is it time to stop talking about appendicitis? No! It is just the time to *begin* talking about appendicitis, and talking most seriously and emphatically about it. When you know that in our best hospitals better than 98 per cent. of all the *acute* appendicitis cases, including those with abscess and peritonitis, are saved, and when you know that scarcely one out of a hundred of the cases of acute appendicitis operated during the first twenty-four hours of the attack is lost, think what the results must be in the other hospitals to make the general average so appalling! There is no palliative excuse for a mortality of 10 per cent. in appendicitis. That rate is simply shocking. Just think! Even the cases of appendicitis with perforation into the free peritoneal cavity have now but a very slight mortality when operated in time by experienced hands; and still the appalling combined rate is 10 per cent., including the chronic and the interval cases,—which should show practically no mortality at all,—as well as the acute cases. It is shocking! It is frightful! These patients did not die because of the operation,—do not misunderstand me,—they died in spite of it. They died in the hospital under a surgeon's management, but they died not so much because of any fault in technic as because of the fact that they did not reach the hospital in time for successful operation. *Procrastination was the cause of death*—the almost criminal cause. The initial symptoms of appendicitis are clean-cut and almost unmistakable. The later symptoms are equivocal and not to be relied on. The mode of onset of an attack of appendicitis is no clue to its probable course or complications. We can never tell in a given case what the next day may bring. *Therefore, operate to-day!* By operation we take the course of the disease into our own hands. By not operating we leave the case in the hands of a blind and often terribly cruel fate.

A mild attack of appendicitis which starts out with colicky pains, nausea and vomiting, and a slight elevation of temperature may develop a leukocytosis and local sensitiveness of the right flank in the first six or eight hours of the attack. By the next morning the pain and temperature may be gone entirely. The doctor then is in a quandary. He is unable to tell from the symptomatology whether the patient is going on to an uneventful recovery because the contents of the affected appendix have drained into the cecum, or whether he is headed straight for the grave because the infected appendix has undergone complete gangrene. A gangrenous appendix causes no pain because its nerves are dead. It produces no elevation of temperature or leukocytosis because absorption of the products of infection are impossible through its dead mucosa. When an apparently mild attack of acute appendicitis has reached such a stage, all the doctor can be certain of is that the patient has appendicitis. *Then* is the time to operate. The disappearance of pain is the last call to operation. If the appendix is gangrenous, the next symptom will be that of a rapidly spreading and probably fatal peritonitis. Remember that the appendix which becomes suddenly completely gangrenous forms no adhesions, and when it ruptures, it empties its contents into a free and unprotected peritoneal cavity. On the other hand, if the appendix is not gangrenous, but is apparently going on to recovery, its removal at this time prevents a recurrence of the attack, much subsequent pain, and the ever-present danger of perforation in a recurrent attack.

I well remember the first case of total gangrene of the appendix which I operated (1892). I had great difficulty in persuading the patient's people to have the operation performed. On the evening of the first day of his attack this man had a fever of 104° F., a very rapid pulse, and the other typical clinical symptoms of appendicitis. When I saw him the next morning the pain and fever had entirely subsided. I insisted, nevertheless, on an operation, because I believed and taught then, as I do now, that every case of appendicitis should be operated, and operated as soon as diagnosed. We took him to the hospital. When we opened the peritoneum the dead appendix shot out into the field, completely black and gangrenous, and without an adhesion around it, but also without pus in the free cavity, because we had operated before perforation had had time to occur.

Since that time I have seen innumerable cases of gangrenous appendicitis. In diagnosing them one must always bear in mind that pain, nausea and vomiting, fever, and leukocytosis are all reactions to infection, not infection themselves. They are not disease. They are the manifestations of disease in the living. The appendix which is dead, like the patient who is dead, presents no symptoms. The living appendix is painful and absorbs the products of bacterial infection, which produce fever and leukocytosis. But the dead appendix has no sensation and no power of absorption. The patient with such an appendix in his abdomen has no symptoms until its necrotic wall ruptures and a spreading peritonitis sets in.

Remember that the mortality rate in appendicitis in children is three or four times as high as in adults. Therefore everything which I have said about appendicitis and its treatment in adults holds at least in triple measure for the cases in early youth. No doctor lets his own child go unoperated through an attack of acute appendicitis. Why should he treat his patients any differently?

We surgeons are not talking enough about early operation in appendicitis. We must stir up those of us who are not thoroughly alive to the facts of the situation. Appendicitis is so common and so important a condition that the indications for its treatment should and *must* be a matter of common knowledge among doctors, laymen, and medical paragraphers.

A DIAGNOSTIC TALK ON INTESTINAL OBSTRUCTION DUE TO A LARGE GALL-STONE

HISTORY

The patient is a woman, aged seventy-seven years. She entered the hospital April 18, 1915, with the following history:

On Thursday, April 15, 1915, she was seized with pains "all over the abdomen." She became nauseated very soon afterward, and vomited. The vomiting was repeated at intervals of about every three hours. She had no chill or fever, but her bowels were constipated. On Friday, the second day of the attack, the intensity of the pain diminished; then it practically ceased, but the patient continued to complain of nausea and vomited. Each succeeding period between the attacks of vomiting became gradually shorter and the vomiting a little more severe in character. On Friday night the odor of the vomitus became offensive and feculent. Friday night the patient vomited five times. She suffered no severe pain, but had a dull ache in the abdomen. On Saturday and Sunday she vomited every two hours or so. She was given calomel, sodium phosphate, and enemas, but without the production of an adequate bowel movement. Then the abdomen became distended. During Saturday night and early Sunday morning vomiting increased in frequency, coming on almost every half-hour. It became almost projectile in character, and retained its feculent odor. The patient had no chill, but developed a slight fever about Sunday noon. She was brought to the hospital late Sunday night.

For the last ten years she has had attacks of colic "all over the abdomen" every three or four months, usually accompanied by vomiting. *She has never been jaundiced.* She calls these attacks "bilious spells." She takes a cathartic for them, and is relieved. She has had no urinary complaint at any time.

Examination of the urine on admission to the hospital showed no albumin or casts. The white cell count was 16,000, and the temperature was 99.2° F. She was given a thorough gastric lavage and a large quantity of offensive material was thus removed from the stomach.

COMMENTS

Dr. Murphy (April 19, 1915): This patient entered the hospital last night, and was operated shortly after midnight. I want to call your attention to the fact that on Friday the patient vomited about every three hours; on Saturday, about every two hours, and on Sunday, every half-hour. That increase in the frequency of vomiting is very suggestive of one form of mechanical ileus—obturation ileus. The doctor says in the history that the vomitus had a fecal odor. I should not use that term. The vomitus was offensive rather than fecal. Only twice in my experience have I seen actual fecal vomiting. It is terrifically offensive. In an intestinal obstruction the gastric contents are first regurgitated. Then, if the vomiting continues, duodenal contents come up, then jejunal contents, and then the contents of the ileum. It is rare that actual formed feces are found in the lower ileum or are able to pass backward through the ileocecal valve from the cecum. Remember, formed feces are found only in the colon, whereas the jejunum and ileum contain fluid intestinal contents normally.

The so-called "bilious attacks" which the patient mentioned in giving her history rather suggest gall-stones; but the complete absence of jaundice militated against this supposition. Could she have had a partial intestinal obstruction extending over a period of ten years of time? I scarcely think so. If it had lasted a year, or six months, or three months, I might think so, but a period of ten years is too long for a chronic stenosing intestinal obstruction. Moreover, the "bilious spells" cleared up entirely after each attack.

There is a type of chronic annular obstruction of the bowel which may run its course over a number of years. I recall one of my early experiences with a case of this kind. I saw the patient first in 1893, at the time of the World's Fair, but she declined operation then. I finally operated her three years later for the obstruction, which had been gradually increasing in severity as time went on. Previous to operation we had her under observation in the hospital for about two weeks. We brought her into clinic a number of times to demonstrate the visible peristalsis and the borborygmus. All one had to do to elicit these signs was to give the patient a full meal of meat; borborygmus could be heard all over the room very soon afterward, and active visible peristalsis accompanied it. If the patient was given only a light meal, no obstruction symptoms at all developed, as a rule. When the borborygmi were active, one could put a stethoscope over the abdomen and locate the exact point where the explosion occurred, which was at the site of the obstruction. Through the abdominal wall we could see the peristaltic wave pass along until it reached the site of the obstruction, and then came the climax and the explosion. Then silence. After a few minutes more, another wave would come along and the series of phenomena would be repeated. One can observe this series of symptoms in most cases of chronic intestinal obstruction if one has not been unwise enough to give the patient an opiate beforehand. Opium stops the peristalsis and thus covers up this important symptom.

When we examined this patient's abdomen last night (Sunday) we found it moderately distended, but not rigid, nor was it soft. There was no visible peristalsis, but one could hear it going on in spite of small doses of morphin that had been given before she came to the hospital. The sounds were best heard low down in the right flank. She complained of no particular pain on admission to the hospital, perhaps partly because she had been given morphin; but she was somewhat sensitive to deep pressure in the appendiceal area, but nowhere else. No attempt was made to give the patient a bismuth meal and then make an *x*-ray examination, because of her great prostration.

At her age we thought, of course, of the possibility of malignant neoplasm of the intestine, but there was no mass palpable anywhere, she had had no previous loss of weight, and there was no history of blood or mucus in the stools. There was no movable dulness in the flanks. Remember that the bowels are as superlatively silent in paralytic ileus as they are superlatively noisy in chronic obstructive ileus.

In spite of the history of a relatively acute onset, we knew that we had to deal with a chronic mechanical obstruction because of the presence of borborygmi.

We felt we could rule out volvulus because that condition is usually accompanied by the presence of a firm, resistant mass in the abdomen, made up of the distended twisted loop of bowel. Besides, although volvulus may be a recurrent affection, its course rarely extends over so long a period of time as that stated in this patient's history—ten years. I have never yet seen a volvulus which did not form a palpable tumor. In one of my cases, a young woman, the volvulus made up a tumor mass as large as a fourteen months' fetus, if you can imagine such a monster. I was called to see the patient on the evening of the second day of her illness. When we opened the abdomen a few hours later, the enlarged sigmoid shot out of the peritoneal cavity, and then exploded, making a noise like the bursting of an air-filled paper bag.

We did not consider intussusception seriously in this woman last night, because intussusception is not a common form of ileus in the aged. It is a lesion of childhood and youth. A Meckel's diverticulum, too, would be expected to make trouble earlier in life than at seventy-nine.

When you see a patient with an intestinal obstruction of four days' duration, the question for you to settle immediately is whether it is a strangulation or an obturation ileus. A four days' old case of strangulation ileus is either dead or practically dead; and strangulation occurs commonly both with volvulus and with bands. One of the first questions which I asked this patient's doctor when he called me up for consultation was, "How are her hands? Are they cold or warm?" If she was already cold, I knew she would die. I had no desire to have her brought to the hospital for me to sign her death certificate. The doctor replied, however, "Her hands are not cold, and neither are her legs and

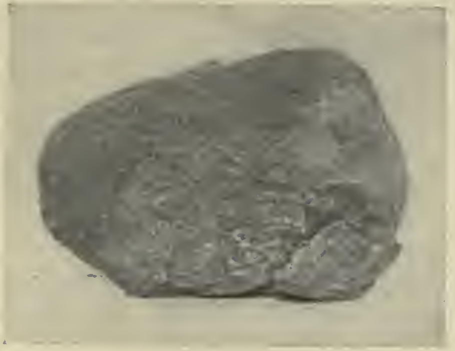


Fig. 125.—A large gall-stone (two-thirds actual size) producing obturation ileus by obstructing the lumen of the lower ileum.

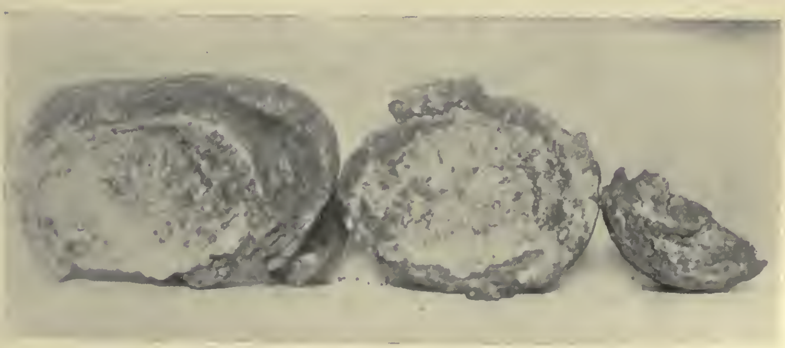


Fig. 126.—Cross-section (two-thirds actual size) of a large gall-stone producing obturation ileus in the lower ileum.

feet, although she has been vomiting recently every twenty minutes or half-hour." Even when I saw the patient, after she had been brought to the hospital, her hands were not cold. Therefore it was evidently not a strangulation but an obturation ileus, and an obturation of itself does not produce an early exitus; death is postponed for six, seven, eight, nine, or even more days, as a rule, unless the obturation is complete and located high up in the duodenum. One not infrequently sees old persons with carcinoma of the large intestine who have not had a bowel movement for ten or twelve days, and yet do not appear to be in an immediately dangerous condition. With the strangulation type of ileus, however, the patient's skin rapidly grows cold and clammy, and he becomes too prostrated to turn over to vomit even, and just spews out the foul-smelling vomitus. When his skin is wet, clammy, and cold, you know he is a candidate, not for the doctor, but for the undertaker.

Beyond the fact that we could pretty definitely state this to be a case of obturation ileus, we could not make a differential diagnosis or locate the exact point of the obstruction, except that the local sensitiveness over the appendiceal area rather pointed to the right lower quadrant as the seat of the trouble. We opened the abdomen by a right paramedian incision below the navel, and a small amount of serous fluid immediately escaped—not enough apparently to have given percussion flatness. We then reached down into the right lower quadrant and picked up the cecum and ileum at their juncture. They were both collapsed. Then we started to run the collapsed ileum upward through our fingers, and we soon found an organic adhesion between it and the bladder; but no ileal angulation appeared to be produced by it, and no loop of intestine had slipped over or under it. This we freed and covered the bowel abrasion with an over and over stitch. Moreover, the intestine immediately above this adhesion was just as collapsed as the intestine below. I followed the ileum still further up until I came to its juncture with the vitello-intestinal duct. Then I could see the cause of the obstruction shining through the dilated intestinal wall, not as clear as crystal, but perfectly evident. There was an enormous dilatation of the ileum above this obstruction, and complete collapse of the intestine below it. What was it? A gall-stone! And here it is—the size of a hen's egg. It is made up almost entirely of cholesterin, and wedged around it was quite a little inspissated fecal material. The stone may have been in transit for years—possibly for the entire ten years that she has been having abdominal symptoms, edging from one pocket in the intestinal wall to another, forming a diverticulum, remaining stationary for a time, and then finally moving on, each time with the production of more or less colic. Finally it became wedged at the narrowest point of the gut, which is at the site of the vitello-intestinal duct attachment, and in this location it produced the present serious symptoms by completely plugging the bowel lumen.

The bowel was split parallel to its long axis sufficiently to permit the mass to escape. The incision was then closed with a Czerny-Lembert suture. An examination was made of the gall-bladder, and it was found free from stones, The dome of the gall-bladder was organically united to the side of the duodenum, showing where the stone had ulcerated through the wall of the gall-bladder and wall of the duodenum into the lumen of the duodenum, thus accounting for the presence of such a large stone without a history of typical gall-stone colic or icterus. This is the route commonly followed by stones of such great magnitude.

I remember a somewhat similar case of obturation intestinal obstruction which I had some years ago in the County Hospital. The patient, when a boy, had eaten a large amount of plum pudding on a Thanksgiving Day some twenty years before. Ever since then he had had recurrent attacks of intestinal colic from time to time, until we operated him. I took out that mass of plum pudding twenty years after he had eaten it. It was almost as inspissated as this gall-stone, and was covered in just about the same way with offensive, dense, fecal material. I always start looking for the seat of an obstruction in about the same way that I did here, that is, from below upward, beginning at the ileocecal valve. It is relatively little trouble to handle the intestines if you begin in this order. The collapsed intestine slips rapidly through one's hands, and when one reaches the dilated loops of bowel, he is at the site of the blocking.

Remember that reflex ileus runs its course without temperature, while a case of septic ileus always has some fever. A mechanical obstruction, too, never has an initial elevation of temperature. A paralytic ileus never has an initial increase in peristalsis sufficient to produce borborygmus. These are milestones in the diagnosis of ileus. In the reflex type of ileus vomiting decreases as time passes, whereas in the mechanical type the vomiting increases with the lapse of time. Leukocytosis is absent at the onset of a mechanical obstruction, but is often present in the later stages, as in this patient here.

Never give a hypodermic for an acute pain in the abdomen until you have made the differential diagnosis, ascertained the cause, and are certain of it. If you cannot make the diagnosis, do not wait two, three, four, or five days. Do something! The one lesion of the abdomen the mortality of which has not diminished in a third of a century is internal intestinal obstruction. Why? Because the diagnosis is not made until the patient is moribund; then a splendid technical procedure is carried out, and the patient dies cured! That is the proposition. Do not wait! Do something! Make the diagnosis! Then go ahead and do at once whatever you are going to do. I think nothing has happened in our National Legislature in recent years which has given me more personal satisfaction than the present Harrison law regarding the use of narcotics. There is just one place for the hypodermic, I believe, and that is in the vault, with the combination forgotten. Then the doctor cannot commit the crime of destroying the symptoms of the disease by a hypodermic injection of morphin before he has made the diagnosis, thus making it difficult or impossible for any one else to make the diagnosis for him. After the differential diagnosis has been properly made, however, the use of the hypodermic is an entirely different proposition.

[NOTE.—The patient came from the operating-room in good condition. Her stomach was again washed out, and it contained a goodly amount of the same offensive material. Her vomiting ceased. Her delirium increased as time passed on; she had proctoclysis with sodium bicarbonate to neutralize her acidosis, and she died twenty-three hours after operation, from her intestinal intoxication, whatever that may mean. This is the classic course of patients with prolonged obstruction, and the danger is especially great when the patients are advanced in years. The absence of icterus is typical of cases in which the stone escapes from the gall-bladder through the wall into the intestine and is one of the important lessons to be learned from this case.—Ed.]

UNSUCCESSFUL GASTRO-ENTEROSTOMY FOR ULCER—AN ANALYSIS OF ITS CAUSES—SUGGESTIONS FOR BETTER TECHNIC

A CLINICAL TALK BY DR. WILLIAM J. MAYO

DR. MURPHY (December 19, 1914): We have a special opportunity to-day which we must not miss. Instead of proceeding with the next case, we shall ask to hear what the experience has been and what the conclusions are of *the* master surgeon in this line of work—not the master surgeon of Minnesota, or of America, or of the Northern Hemisphere, *but of the world!* Our next patient has had a gastro-enterostomy performed elsewhere for what was said to be a gastric ulcer. Now she has not only a return of all her former symptoms, but, in addition, has had some attacks of hematemesis recently. We want to know what is best to do for such patients, and we want the man who best knows to tell us. Let me present to you—not introduce, for medical men everywhere know him—Dr. William J. Mayo. He has done more to standardize and advance surgical procedures in the upper abdomen than any other surgeon living or dead.

DR. W. J. MAYO: *Gentlemen:* I assure you that I am proud of the opportunity Dr. Murphy has given me to appear in these Clinics, which are so widely and favorably known and so unlike anything that has ever been done before in the field of clinical surgery. Dr. Murphy is preëminently a clinical man. In seeing his work one is impressed with the fact that he has gone far into various fields of special work, and, as may be said of all great general surgeons, through this wide experience has been enabled greatly to advance the several types of special surgery. I, like many others, have been an admirer of Dr. Murphy, and have followed his work for many years. It scarcely need be said that his fame as a surgeon is due not alone to the Murphy button, but to his progress along general scientific lines. I well remember the time of the Haymarket riot, when he sutured abdomens and intestines and acquired the name of "Peritoneal Murphy." Let me call attention to the master oration, delivered at Denver seventeen years ago, on "The Surgery of the Lung," which has served as a basis for surgical work done on the lung since that time; and again to his work on "The Surgery of the Bloodvessels," "Surgery of the Nervous System," and "Surgery of the Joints." I mention these points to emphasize the great advances in surgery that have come from the general surgeon whose experience has been extensive in special fields.

In looking back over the development of diagnostic methods we find a rather definite division into four periods: The first period might be called that of clinical assertion, when a statement was made but not proved either at the operating-table or at necropsy. If the man making the statement was sufficiently distinguished, and held a responsible or important position, his words were treated with great consideration, and too often passed into text-books on medicine, not as mere assertions, but as accepted facts. It has been most difficult to eradicate the effect of such statements once they have gone on record in books.

Following this period of diagnostic "assertion" came the second, or postmortem, period, which we passed through, accepting nothing as authentic which could not be demonstrated at the postmortem examination. This was a valuable foundation to build upon, but a poor superstructure. The postmortem does not necessarily reveal the disease the patient suffered from during life, but merely the disease from which he died. Nothing can be more deceptive than postmortem findings unless it is statistics.

From the postmortem we came to the third, or the experimental, period. Active, well-trained men devoted themselves to research; nothing was held as true unless it had been proved by experiments on the dog. Many valuable and interesting facts were demonstrated during this period.

The three foregoing periods prepared us for the fourth—that of clinical research, the period which Dr. Murphy has so ably represented in his operations to-day. The most valuable contributions now being made to science are in the form of clinical research, a branch of medicine built on truths which developed during the preceding periods.

The sense of sight has been the acid test in diagnosis. We could be sure only of those things which were demonstrable to the eye. The cavities of the body opened to sight supplied us for the first time with exact knowledge of actual conditions. The Röntgen-ray and other means of sight demonstration have also brought marvelous aid to diagnostic measures. The growth of our knowledge of ulcer of the stomach and duodenum illustrates these facts to a remarkable degree.

Dr. Murphy has asked me to speak particularly on the occasional failure of operation to cure gastric and duodenal ulcer. I believe these failures should be divided into several groups. As is quite appropriate in Murphy's Clinic, I will begin with a hibernianism and say that I think the most common cause of failure to cure gastric and duodenal ulcers by operation is that the patient did not have the ulcer, but did have the operation. This may be explained as follows: The postmortem has given us certain data concerning gastric and duodenal ulcers; nevertheless, our opinions concerning ulcers have been largely based on statements of men distinguished in medical circles, and we have not been able to get rid of these impressions. As a result, the patient who presents himself with a certain train of symptoms is said to have gastric ulcer. On the operating-table the condition is not demonstrable, yet a gastro-enterostomy is done. So true is this that in our own clinic we have cut off somewhat more than a hundred gastro-enterostomies where no evidence of ulcer was discovered at the second operation. To-day we are cutting off few gastro-enterostomies because of the fact that the pylorus is mechanically closed at the primary operation. But there can be no doubt that unnecessary operations are being done. The change in the pyloric tissues by the blocking procedure is such that at the second operation one cannot tell whether the scar is due to the closure or to an ulcer. This might be called the "prepared-in-advance alibi," since it establishes false testimony. The anatomic defect is exceedingly useful in letting the original surgeon slip out from the responsibility of making a gastro-enterostomy the patient did not need. If there are signs of impending perforation or hemorrhage, closure of the pylorus is useful to a limited extent, but as a routine it is, to say the least, unnecessary.

In the second group of cases a stitch ulcer will be found which is due to the use of continuous silk or linen sutures. Permanent silk or linen sutures used in gastro-enterostomies have been removed as long as three years after they were put in. Often after a number of months the infected sutures pass out, but the margins of the gastro-enterostomy are left thickened, with interposing scar tissue and adhesions of the upper jejunum to the transverse mesocolon in the vicinity of the gastro-enterostomy. If this happens, the patient will have a recurrence of all the symptoms of the original ulcer. While true physiologic jejunal ulcers are occasionally seen, the large majority of so-called jejunal ulcers are in reality gastrojejunal stitch ulcers.

I think it may be put down as a fact that if a patient really has an ulcer and an operation based on modern principles is done, the percentage of failures will be very small. Because of deforming scars, adhesions, etc., there will be a higher percentage of failures to cure ulcer of the stomach than of the duodenum.

Failures due to the improper selection of cases or bad technic have led to the erroneous belief that every patient operated on for gastric or duodenal ulcer should be subjected to a long course of medical treatment following the operation.

This brings us to the third group of cases, in which failures to cure may be due to development of carcinoma in gastric ulcer. The older statistics based on postmortem findings show that the percentage of cases in which carcinoma develops in gastric ulcer is very small, but when the cases are studied from the standpoint of clinical and pathologic research, the percentage of cancer degeneration is found to be very large. This is shown in the actual pathologic condition of the excised specimen.

A most important paper, read by von Eiselsberg at the London meeting of the Clinical Congress of Surgeons (August, 1914), showed that 10 per cent. of the patients he had operated on for peptic ulcer died subsequently from cancer of the stomach. In analyzing his statistics we find that 13 (28 per cent.) of a total of 41 deaths occurred from cancer. There were only 23 in which the actual cause of death was shown by postmortem examination. When the cancer risk is considered in addition to the risk and disability due to the ulcer itself, we can only conclude that ulcer in its chronic form is a surgical malady.

Of all the different symptoms produced by ulcer, hemorrhage is the one which operation most often fails to cure. Unless the hemorrhage is preceded and followed by symptoms of ulcer, the probabilities are that the hemorrhage is not due to an ulcer, but to infectious emboli, gastrotoxic in origin, from some other place in the body.

Looked upon as a whole, I think nothing in the field of surgery has given such good results as the operative treatment of ulcer of the stomach and duodenum.

FRICTION BURN OF LEFT ANKLE—CLOSURE OF THE DEFECT BY A PEDICLED FLAP OF SKIN AND FAT

HISTORY

The patient is a man, aged twenty-five years. His present trouble dates back to June 15, 1914. At that time the patient, while working in a mine, caught his left trouser-leg in a moving shaft, pinning the ankle to the shaft and bringing the patient's body against a nearby moving belt. The patient's ankle suffered a severe friction burn on its outer side, about three inches above the joint. This was a third-degree burn. There were second-degree burns on both thighs, on the abdomen, and on the penis, the result of friction with the moving belt. These second-degree burns all healed within a period of about four months, after having discharged serum and pus rather freely during that time. The burned ankle was dressed first with moist and then with dry applications, but has not healed up to the present time. The patient cannot bring the heel of the left foot fully down to the floor, because of the scar tissue which has already developed, nor can he adduct or extend the foot beyond a slight degree.

COMMENTS AND OPERATION

Dr. Murphy (December 23, 1914): The history states the proposition before us fairly well. The friction burn on the ankle was the result of contact with the moving shaft, not with the belt. How to cover over this deep defect so that it will heal is something of a problem. To heal it without a transplant is impossible. The ulcer might be covered with a Thiersch graft; and, no doubt, the Thiersch transplant would live, but just as soon as the patient would return to his work after a Thiersch transplantation the newly planted pieces of skin would rapidly drop off or disappear.

The defect caused by the erosion extends through the skin, fat, and fascia, the entire thickness of the lower end of the fibula, and into the tibia itself for a distance of ⅓ inch. One of the principal reasons why the wound has not yet healed is because its margins are so tightly fastened to the neighboring rigid structures that the wound cannot contract as scar tissue forms and thus gradually obliterate itself. The firm attachment of the wound margins to the fibula and the tibia prevents any extensive contraction of the defect. Further waiting has no value whatever. The defect must be covered in some way, otherwise we shall have to amputate the foot.

We should like to take the graft from the other leg, but we cannot do that because the other leg has not enough unscarred skin available to fit into the defect. The proposition would be relatively easy if we could only throw one leg over the other and make the skin-graft to the left ankle from the right. Because of the large size of the defect which must be filled we shall be forced to get our tissue from the opposite thigh, instead of from the leg, as we should like to do.

Here is the area on the right thigh just above the outer side of the knee where we are going to obtain the pedicled flap for transplantation into the left ankle. [Dr. Murphy outlines the flap with his finger.] When we have dissected back the flap and have sutured it into place over the left leg defect, we shall have to apply a plaster cast to the right thigh and left foot to hold them both firmly together until the graft heals in place.

After we have dissected up the flap there will be quite a large defect left in the thigh surface. For the greater part we can probably draw the edges of this wound together. Even if we cannot draw them entirely together, there is still enough soft tissue left on the surface of the wound so that granulation tissue will form freely, and therefore we can readily apply to the raw surface a Thiersch graft which will adhere and grow.

It is now about six months since this burn occurred. The longer one waits in such a case, the less favorable the case becomes for operation, because of the scar tissue which develops in the base of the defect. There has been too much waiting already in this case, and we therefore have to face conditions somewhat unfavorable to success. It may be that after we have transplanted our flap to its new location new vessels will form very slowly, and that when they have formed, the scar tissue present will contract down still further and strangulate them so rapidly that the transplanted flap will not live. That is a risk which we must run; but there is no other chance of saving this young man's foot.

Notice that in the preparation of this operative area we are not using iodin. We do not use iodin in our skin-graft cases because it blisters so readily, and blisters are frequently followed by superficial purulent infection. One cannot achieve success in plastic work in the presence of infection.

We are applying this plaster cast above and below the flexed left knee in order to assist us in the operation. These figure-of-8 turns which we are making will keep the left knee bent at just the right angle to bring the left ankle near the right knee, and therefore make our operative manipulations less difficult by keeping the parts concerned fairly closely approximated while we are working.

Please note that we have placed no constrictor on either limb. The object of an Esmarch is to stop hemorrhage temporarily. What we are interested in here is permanent hemostasis; and, by not using a constrictor, clotting within the smaller vessels will occur earlier than if we should use one. We are very anxious to have all hemorrhage stop by the time that we cease operating. Should hemorrhage continue, the bleeding will occur underneath the transplant, where a hematoma is likely to endanger seriously the life of the flap. If the flap lives, we shall next consider the proposition of making the patient a new peroneus and tibialis anticus tendon from a strip of fascia. If the flap dies, however, amputation will probably be our sole further recourse.

I am first freshening the cicatricial surface of the ankle ulcer, and am freeing the edges of the wound from their attachment to the bone by cicatricial tissue. Now that we have this area

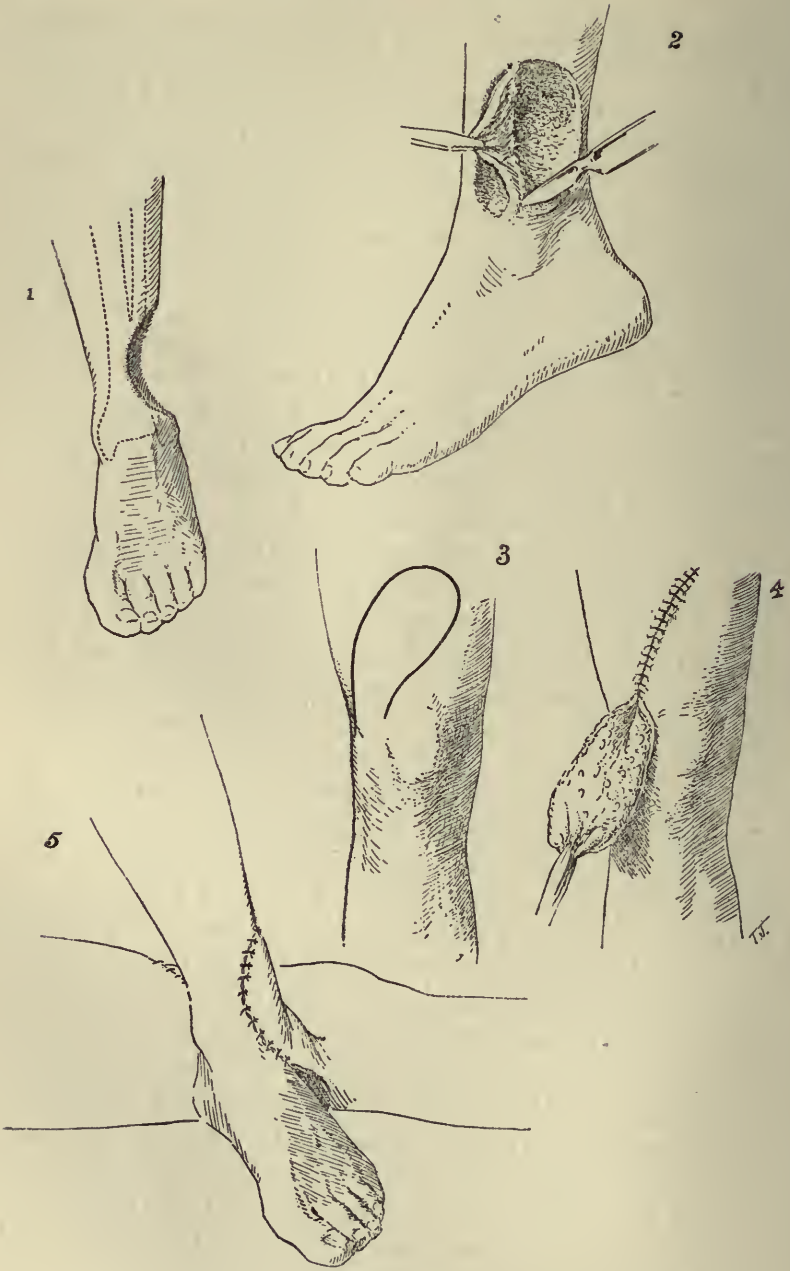


Fig. 127.

prepared, we shall cover it over with a compress wet with salt solution, and wait until the bleeding stops before putting on our pedicled transplant. While we are waiting for the hemorrhage to stop we shall get our transplant ready.

In preparing the pedicled flap we are taking advantage of the richness of the circulation in the vicinity of the knee by placing the pedicle of the flap where it will secure a liberal vascular supply. (See the accompanying illustration, Fig. 127.) We are also careful to plan the flap so that there is no likelihood of a circular scar being formed in this vicinity. Circular scars, as you may know, are often very vicious in their ill effect on the nutrition of a limb. For the flap, we are taking the entire thickness of the skin and subcutaneous tissue of the thigh clear down to the fascia lata. The scar which this flap will leave will not lie over any bony prominence, and therefore should cause the patient no particular inconvenience in any way.

Now that the flap is prepared, we shall wrap it also in gauze moistened with normal salt solution and wait for hemostasis to occur while we suture up the defect which its removal has left in the skin of the right thigh. I am making an accurate apposition of this skin with a series of interrupted silkworm-gut stitches. See! it comes together without any great difficulty. We are going to have a good apposition here, I believe, and shall not need any subsequent Thiersch grafting.

Fig. 127.—Friction burn of the left ankle. Plastic closure of the defect with a pedicled flap of skin and fat from the right thigh.

1, Shows degree of destruction which penetrated through one-third of tibia, and severed fibula completely. The ends of the fibula had united to the sides of the tibia by osseous bridges. The burn covered one-half the surface of the leg, and could not contract because of the fixation to the bone of the scar tissue over the entire area.

2, Dissecting off the granulation and scar tissue at its base down to the normal tissue in the soft parts and to the bone.

3, Showing the outline, size, and direction of the pedicled flap as freed above the right knee.

4, The flap, elevated and denuded area accurately approximated by suture.

5, Shows position of left foot as fixed and held by a plaster cast, so that it could not move from its relation to the right thigh. Also shows the line of suture of the flap to the left ankle defect.

It is now time to turn the flap into the prepared cavity waiting for it. Note that it comes right into apposition where we want it without any appreciable tension on the pedicle. We must fasten it to the wound margin with a series of interrupted stitches. We always put the knots of these stitches on the skin side. If that flap is reasonably fair to us, it will heal in without accident, because we have adjusted it accurately in place without tension on its pedicle. That is all we can do. The rest is dependent on nature's healing powers.

The cast which we applied around the patient's left knee in order to flex it to an acute angle, so that the left ankle would fit close to the opposite thigh, will be continued downward to inclose the area which we have covered with the dressing.

The red bismuth subiodid powder which we applied to the wound before putting on the dressing has no other purpose than to keep the wound dry. Where there is no moisture, bacteria will not grow.

We shall put a weight-carrier of some sort under the left knee, to keep the weight of the limb off the flap and the left ankle, then there can be no undue traction on the pedicle. The knee with its inclosing cast will be suspended on a bar passed lengthwise over the bed. The patient will need the sympathy of every one, I think, between now and a time two weeks from now, because there will be some traction on the pedicle, in spite of all we can do, and it will be sufficient to give him considerable pain, even if it is not enough to compromise the vitality of the flap in any appreciable degree.

[NOTE.—The flap healed in by primary union. The pedicle was detached over half its area on the thirteenth day, and completely separated on the fifteenth day. The area was completely covered with normal skin containing a pad of fat ½ to ¾ inch thick. Through this flap we can readily pass the new tendons so that extension of the foot will be restored.]

A SERIES OF DRAWINGS ILLUSTRATING DR. MURPHY'S METHOD OF SUTURING A PEDICLED MUSCLE FLAP INTO THE LAMINECTOMY DEFECT TO PROTECT THE EXPOSED DURA AND OBLITERATE THE DEAD SPACE WHICH WOULD OTHERWISE FILL WITH BLOOD-CLOT. AFTER INTRADURAL OPERATIONS THIS FLAP TRANSPLANTATION DIMINISHES THE DANGER OF INFECTION AND HELPS PREVENT THE ESCAPE OF CEREBROSPINAL FLUID THROUGH THE WOUND.

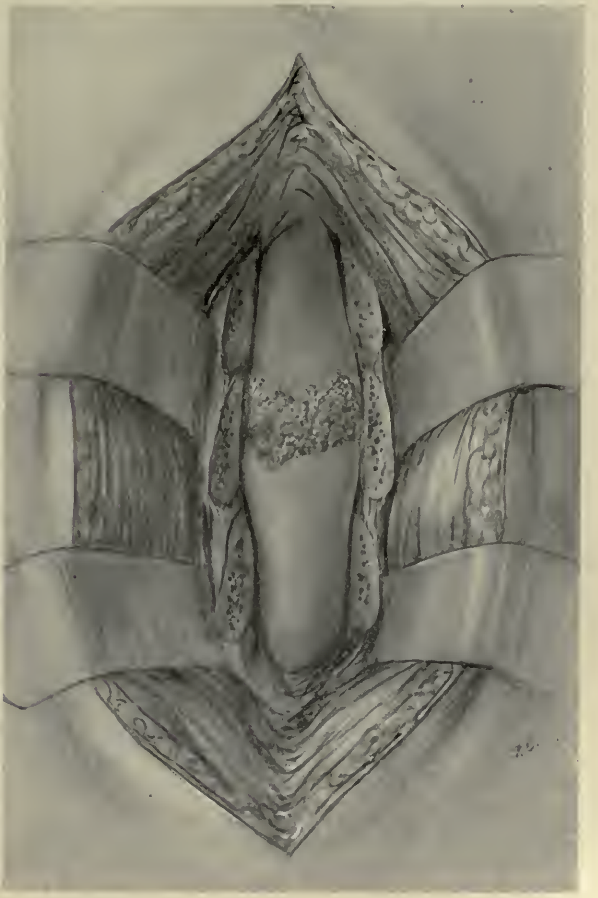


Fig. 128.—Extradural granuloma (tuberculous?) producing symptoms of pressure on spinal cord. Dura with overlying granuloma exposed by a decompressive laminectomy.

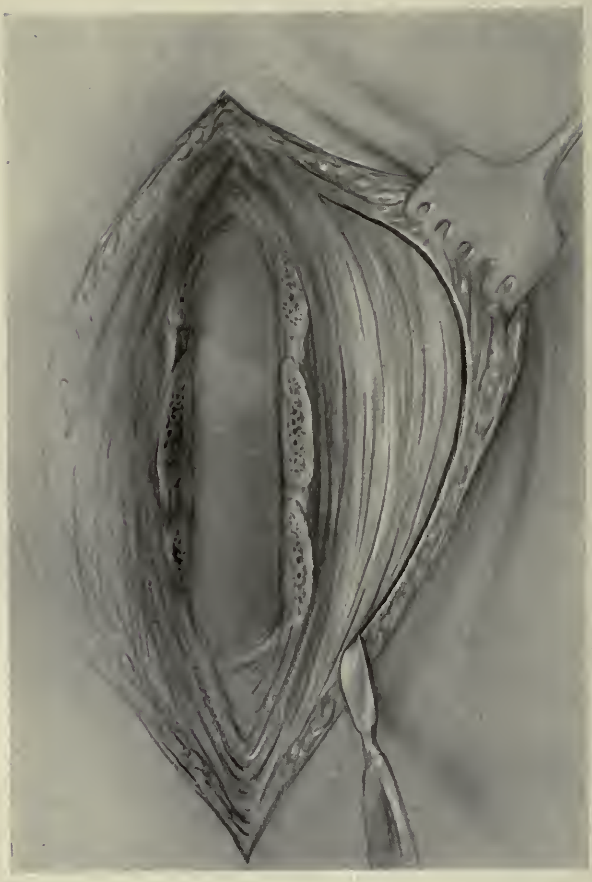


Fig. 129.—Granuloma dissected out so far as accessible. Outlining the pedicled muscle-flap.

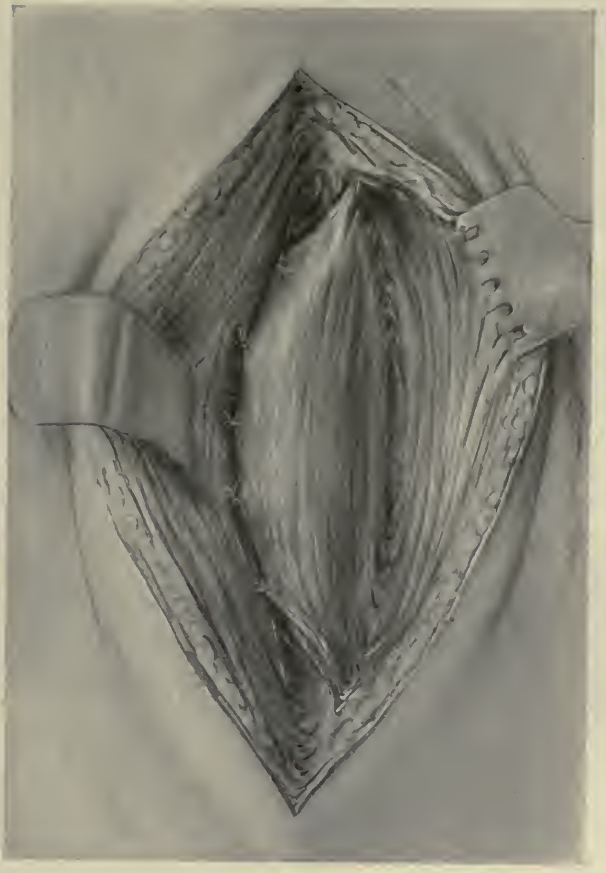


Fig. 130.—Muscle-flap sutured into place across the laminectomy defect, obliterating the dead space and protecting the cord.

EMBRYONIC TUMOR OF THE TESTICLE—EXCISION OF TUMOR AND TESTICLE

HISTORY

The patient is a married man, thirty-three years old, a physician by occupation. He entered the hospital February 4, 1915. There is no history of carcinoma or any malignant tumor in his family. One aunt suffered from a cervical lymphadenitis and died subsequently of pulmonary tuberculosis.

As a child, the patient had measles, whooping-cough, and mumps, without complications. In 1900, fifteen years ago, because of some nasopharyngeal trouble, the patient had his tonsils partially removed, and a little later his right middle turbinate and a polyp of the left nostril. In 1907, eight years ago, the patient had an attack of diphtheria complicated by an acute myocarditis. While recovering from the diphtheria he developed an acute appendicitis. He was not operated on, however, as his heart was considered to be too seriously involved to stand the strain of an operation. Somewhat later in the year, however, when his myocarditis had cleared up, his appendix was removed. He made an uneventful recovery from this operation, except that he developed a stitch abscess after it.

In 1911, four years ago, he was operated for hemorrhoids. In July of the same year he had an attack of what was called acute colitis. His symptoms at that time were repeated chills, fever, griping pains in the abdomen, with mucous stools. He was in bed several weeks with this illness. In 1914, one year ago, he had an attack of acute tonsillitis. His fever was slight at the time, and the illness lasted only about ten days. It did not incapacitate him for work. Since then he has had several mild attacks of tonsillitis. With these attacks he usually notices some acute enlargement of the regional glands in the neck. He has noticed for some little time that his uvula appears elongated.

The patient's present trouble, because of which he comes to the hospital, began in September, 1914. Abnormal frequency of urination was the first symptom noted. This frequency was more noticeable in the daytime; but at night he usually had to rise once or twice, whereas previously he had practically never needed to. A month later, in October, 1914, he noticed that he became tired very easily. He had no fever at this time, however, and his appetite was unimpaired. In November, 1914, he noticed for the first time a hard nodule, about the size of a hazelnut, in the left epididymis. He discovered this lesion quite by accident, and not because it caused him any inconvenience. The nodule has gradually increased in size since then, until it is now about twice as large as when first discovered. The patient's left testicle has always been smaller than his right, until this nodule appeared in the former. Now the left appears a little larger than the right.

The patient, being a physician, naturally feared that he was developing an epididymal tuberculosis. The history of tuberculosis in his family furnished additional ground for this fear. Accordingly, about the middle of December, he performed a von Pirquet tuberculin test on himself. As a result of this test his left arm became considerably inflamed, and the axillary and the epitrochlear glands became enlarged. The diagnosis of tuberculosis thus becoming probable, he had a subcutaneous injection of Koch's old tuberculin made to confirm it. Accordingly, on January 5, 1915, he was given 5 minims of No. 2 dilution of old tuberculin at 3.30 P. M. He had a chill that night, and twelve hours later developed a temperature of 102.5° F. He was sick in bed for four days. For three days he had fever, general malaise, night-sweats, and increased frequency of urination. *There was no testicular reaction*, however, no tenderness or swelling of the suspected nodule. [NOTE.—This was not a typical reaction, therefore, notwithstanding his fever.—ED.]

For the past month (January, 1915) the patient has been very busy and on his feet a great deal. In consequence, he has experienced considerable dragging discomfort and pain in the scrotum. He has worn a scrotal supporter since November, 1914, but it does not seem to relieve his present pain and discomfort. In the standing position there is considerable engorgement of the scrotal veins (varicocele). This engorgement disappears on lying down. For a long time the patient has had some tendency to constipation, but it has not been so marked since his hemorrhoids were excised four years ago—in 1911.

Three weeks ago the patient put himself on tuberculin treatment, beginning with 5-minim doses of dilution No. 4. The next week the dose was increased by 2 minims, but still there was no constitutional reaction. Last week he took 9 milligrams of dilution No. 4, and developed a temperature of 99.4° F.

He positively denies venereal infection of any variety.

COMMENTS AND OPERATION

Dr. Murphy (February 6, 1915): The most frequent early symptom of genito-urinary tuberculosis is increased frequency of urination, whether the tuberculosis is primary in the kidney or in the testicle, and the severity of the disturbance usually increases as the tuberculosis advances. Dysuria may be present even when renal tuberculosis is confined to the cortex of the kidney, and when no tubercle bacilli are passing down the ureter, so far as can be determined by microscopic examination of the urine or by animal inoculation. I presume that in such cases the irritation of the neck of the bladder must be due to the toxins excreted in the urine. Likewise, in tuberculosis of the epididymis there is usually increased frequency of urination even before any pus appears in the urine.

The presence in the urine of an increased number of leukocytes is commonly the next symptom to appear. Then, in order of frequency, comes hemorrhage, probably. Sometimes hemorrhage is the initial symptom of genito-urinary tuberculosis, particularly of the kidney, whether primarily in the cortex or in the pelvis.

When tuberculosis begins in the cortex of the kidney, the most common location of onset, increased frequency of urination is generally the first symptom. The number of leukocytes in the urine is usually increased, but seldom, if ever, to a degree which would enable one to call it a pyuria. When the cortical lesion burrows its way to the pelvis and then ruptures through the mucous membrane, there is a discharge of a considerable quantity of caseous material or pus, and, often, blood. It may be that the hemorrhage from this rupture into the pelvis is the first symptom of the disease noted by the patient. When tuberculosis begins in the mucous membrane of the pelvis, pyuria, hematuria, and dysuria commonly appear simultaneously.

When the tuberculosis is primary in the epididymis, increased frequency of urination is usually the first symptom, and generally antedates the discovery of the nodule by the patient, as was the order in the present case. We have had placed on the board a chart indicating the relative frequency of tuberculosis in the various portions of the epididymis and in the testicle. (See CLINICS for February, 1914, page 67.)

In tuberculosis the epididymis and the testis are often so closely grown together and infiltrated with the granuloma that it is difficult or impossible to tell what is primary and what is secondary deposit. But it is possible to record, when the scrotum is opened, which portions of the epididymis and testis are found involved and which are free from the disease. This chart tabulates the results of such a study, carried out on specimens obtained by operation and by autopsy. Note that the globus minor was found involved in 100 per cent. of the specimens.

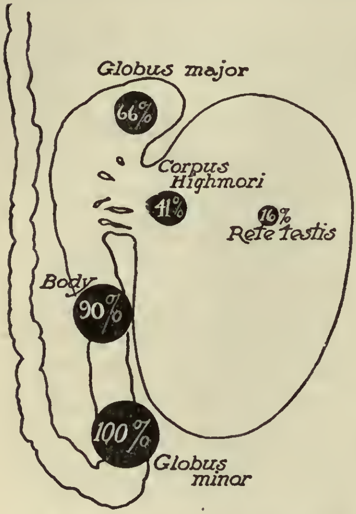


Fig. 131.—Diagram showing the relative frequency with which different portions of the epididymis and testis are found affected by tuberculosis at operation and autopsy. These figures include not only primary, but also secondary, involvement. Note that in every case the *globus minor* was involved either primarily or by extension. Note how seldom tuberculosis begins in or extends to the rete testis—only 16 per cent. of the cases.

This observation does not mean that the disease was primary in the globus minor in 100 per cent. of cases, but that in every single case examined the disease *involved* the globus minor either primarily or secondarily. The body of the epididymis was involved in 90 per cent. of all the specimens, whereas the globus major was involved in only 66 per cent., and the corpus Highmori in 41 per cent. The disease originated in or extended to the rete testis in only 16 per cent. of the cases. In how many of the specimens the tuberculosis was actually primary in the testis we do not know from these statistics; but the figure must be very much less than 16 per cent., possibly 5 or 4, or possibly even less. The one pathologic truth to which we can cling fast as the result of this statistical study is that the testis, even in advanced tuberculosis, is rarely much involved by the disease. Primary tuberculosis of the testicle is seen, I believe, practically only in general miliary tuberculosis, where many organs are involved in the miliary spread of the disease. Both primarily and secondarily it is the hilum of the testis which is chiefly involved, particularly the testicular ends of the vasa deferentia.

This pathologic study furnishes us the basis for our surgical treatment. Since tuberculosis is evidently a lesion of the epididymis almost exclusively, our aim should be to remove the diseased epididymis at the earliest possible moment and leave the intact testicle, or, in the infrequent cases where the testicle is somewhat involved, to leave that portion of it which is still undiseased. Thus we shall preserve its internal secretion without subjecting the patient to much additional risk of local recurrence. Moreover, we thus succeed in persuading the patient to the operation at a much earlier stage of the disease than we do if we offer the patient merely castration. I believe that, because of the large number of early operations obtained, the ultimate surgical results of partial excision will be equally as good as or even better than those of castration.

The circulation of the epididymis is separate from that of the testicle, and, therefore, the epididymis can be excised completely without interfering in any way with the testicular circulation. (See the CLINICS for December, 1914, page 1192.) It is just as easy to remove the diseased epididymis without disturbing the integrity of the testicle as it is to excise the appendix without damaging the caput coli. Once the epididymis is attacked by tuberculosis, its function as a transmitter of the external secretion of the testicle, the semen, ceases. No further damage can be done by excising the diseased organ, the epididymis, and a further extension of the disease, locally in the testicle and distally in the seminal vesicles, the prostate, and the bladder, can be prevented.

This patient, being a physician and well informed, has come to us early in the course of his disease. He also has readily consented to the operation. He has given this subject the thorough study which his own deep personal interest in the disease inspired, and he has arrived at the same conclusion as we.

Visiting Doctor: Why was there no local reaction in the testicle at the time the tuberculin injection was made?

Dr. Murphy: That is a question which we should like to have some one answer for us. The patient is convinced that his lesion is a tuberculosis of the epididymis. I am not certain that it is tuberculosis: there are certain unusual features here which are difficult to explain, one of which is the absence of the local tuberculin reaction. I agree this far with the patient, however, that his is a surgical case, and that operation is needed, probably urgently.

The nodule is located at the upper pole of the testicle. Is it in the epididymis or in the testicle itself, or does it involve both? I am rather more inclined to believe that it lies in the upper pole of the testicle, for I think that I can feel the globus major of the epididymis running over it. The patient, however, thinks that the soft structure which we feel there is a varicosity. I do not think so. If the nodule is located in the globus major, it is tuberculosis in all probability. If it is located in the testicle, it is not tuberculosis. If the nodule is a sarcoma, carcinoma, or embryoma, it is just beginning.

Remember, tuberculosis is not so common in the globus major as it is in the globus minor. It is 50 per cent. more frequent in the latter than in the former. On the other hand, the globus major is the favorite location for cysts of Morgagni and for sper-

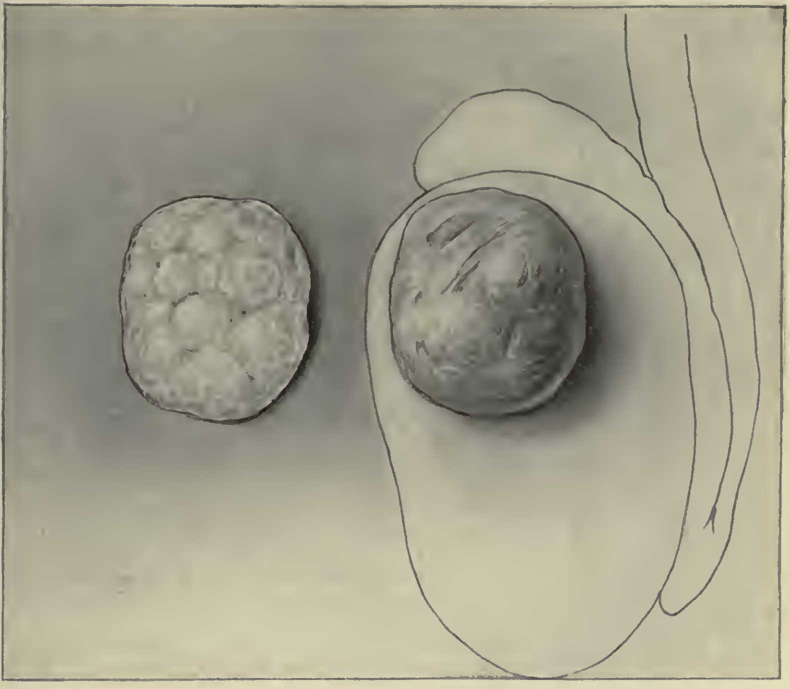


Fig. 132.—Embryonic tumor of testicle. The drawing shows the location of the tumor in the testis and its gross appearance, both externally and on cross-section. Note the well-developed fibrous capsule.

matocele. I do not believe that we shall find a gumma. The patient denies venereal infection; he has no evidences of a present or past lues; and he feels so certain of never having had syphilis that he declined to undergo the expense of a Wassermann reaction.

Some of you may remember that a few months ago we had a somewhat similar case of tumor of the testicle. (See CLINICS for December, 1914, page 1197.) The patient, the brother of a physician, was so certain, as was also the doctor, that the patient had never had syphilis, that a Wassermann test was not done in that case. Moreover, the luetin test was negative, and there were no clinical evidences of syphilis in the patient or in his family. Nevertheless, the operation revealed the tumor to be a large typical caseous gumma, the specific nature of which was unmistakably evident, both grossly and microscopically. The Wassermann test subsequently made was reported "triple positive."

The syphilitic tumor of the testicle often shows sudden increase or diminution in size, due partly to vascular and inflammatory changes in and about the granuloma, and partly to acute hydrocele, which comes on rapidly and as rapidly disappears. There is nothing of that nature in this patient's history. His growth has continued to increase in size regularly and gradually. I am confident that we do not have to deal with a gumma in this case.

[Dr. Murphy made the usual oblique incision over the lower inguinal region and delivered the testis and epididymis through the cut.]

It still is not easy to say whether the mass lies in the globus major or in the upper pole of the testicle, for as the globus major is adherent to the upper testicular pole, I cannot readily separate it from the latter. The globus minor lies entirely free, just as we decided before operating. The globus major is attached as closely and as firmly to the upper testicular pole as if by inflammatory adhesions.

We shall first dissect free the globus major and find out what lies beneath it. The artery to the testicle runs down on the inner side of the epididymis and testicle, and the artery to the epididymis on the outer side. Therefore, our line of dissection will lie along the outer side of the testicle, and we shall turn the epididymis upward and inward as we proceed to free it. As we complete the separation at the inner line of cleavage between the epididymis and the testicle we shall proceed carefully with our dissection, so as to avoid injuring the testicular artery. . . .

Now that the globus major has been dissected free, we can see that the disease lies practically entirely within the tunica albuginea, that is, within the testicle. Whether the disease began in the globus major or not we shall soon know. Present appearances are against this origin. It looks now more as if a pathologic process starting from the upper pole of the testicle was spreading through the tunica albuginea and involving the globus major, forming adhesions between the two structures. It does not look like tuberculosis. This is not the way tuberculosis spreads.

I am making a circular incision through the tunica albuginea at the lower level of the nodule, thus excising the entire upper pole of the testicle. There seems to be a well-defined capsule about the nodule, and I am working close to that in my dissection. I shall not open the nodule until I have enucleated it completely. . . .

I have it out now. Let us split it down the middle. The cut surface does not look like tuberculosis—the capsule is too well formed, and there is no caseation in the center. It is light gray in color and is somewhat softer in the center than at the periphery, but this central change looks more like the ischemic necrosis of a tumor with a rather sparse blood-supply than it does like the caseation of a tuberculoma. It looks still less like a gumma than like a tuberculoma. There is not the inflammatory zone and the granulation tissue at the periphery of the lesion which one sees in gumma, and gummas of this size do not often become so soft in the center. In spite of necrotic foci they usually remain quite hard because fibrosis ensues so rapidly.

We are sending half of this tumor to the pathologist for a frozen section, and the other half we shall pass around the clinic,

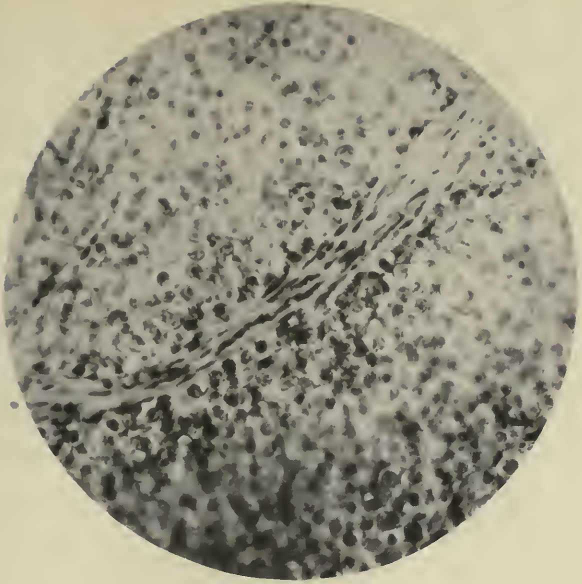


Fig. 133.—Embryoma of testicle. Note the capillary running through the center of the field, about which are grouped the rapidly proliferating (many mitotic figures) epithelial cells derived from the epiblastic layer of the embryoma.

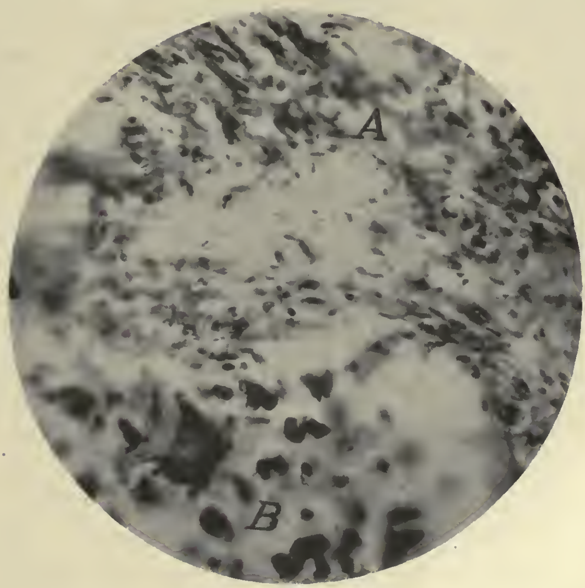


Fig. 134.—Embryoma of testicle. This high-power photomicrograph shows spindle cells (A) (mesoblast) in the upper two-thirds of the field, and epithelial cells (B) (epiblast) in the lower third. Note the sharp differentiation here between the two embryonic layers, which, as a rule, tend to become inextricably mixed.

so that you may all observe its macroscopic appearance. If it is a sarcoma, it has an unusually well-marked capsule for such a tumor, and yet I cannot give a very good prognosis if it be a sarcoma, because in spite of the capsule the growth appears to have penetrated the tunica albuginea and has already involved the surface of the globus major. Remember, this man had no regional adenopathy, and regional adenopathy occurs rather early, as a rule, in both carcinoma and sarcoma of the testicle. If the nodule should prove to be a tuberculoma or a syphiloma, the prognosis will be good, and we shall leave in the epididymis, which has not yet been appreciably involved in the lesion. If it proves to be a sarcoma, the prognosis will not be so good, not only because of the likelihood of recurrence *in loco*, but because a sarcoma of one testicle is very apt to recur in the testicle of the opposite side. Remember that a sarcoma of one kidney, even when excised, is practically always followed by development of a sarcoma in the opposite kidney; and that a sarcoma of the ovary, after removal, is very frequently followed by the development of a sarcoma in the other. The same bilateral involvement often occurs with sarcoma of the testicle, although not so frequently as with sarcoma of the kidney or ovary.

Dr. Sweek (the pathologist): The microscopic appearance of the nodule is not very characteristic. There is some necrosis and necrobiosis in the center of the nodule, but no typical caseation, and no tubercles anywhere. I have not seen any giant-cells in the sections which I have examined, and there are none of the endarteritic changes seen in a syphiloma. There are many areas of deeply staining round-cells with karyokinetic figures. I think this is most likely a round-cell sarcoma.

Dr. Murphy: If this is a round-cell sarcoma, the patient should have not only the rest of this testicle and epididymis excised, but also the regional glands and possibly the testicle on the other side. We have no permission for so extensive an operation, and I believe it no more than right that the patient should be allowed to express his own wishes on so vital a subject to him as his unsexing. The additional delay of a few days will, I believe, be a matter of relatively slight importance now that the tumor itself has been excised. The pathologist, besides, does not seem too certain of his own diagnosis. Perhaps the paraffin section, which we always make in these cases in addition to the frozen section, may tell a little different story from what we now hear. Certainly we hope that such may prove to be the case. We shall leave the rest of the testicle and the epididymis just as it is, covering up the raw surface of the testicle with the tunica vaginalis. We have now returned the scrotal contents to the scrotum, and are top-sewing the skin wound. That step completes the operation.

[The usual copious scrotal and inguinal dressing was then applied.]

The pathologist's report seems to rule out completely the possibility that this nodule may have been tuberculous. Why, then, did the patient have dysuria, and the marked febrile reaction to the injection of old tuberculin? Certainly, the possibility of renal tuberculosis must be seriously considered, and the patient must be further observed with this possibility in mind. Remember that a tuberculous focus in the kidney, particularly a cortical focus, may undergo spontaneous healing and encapsulation in favorable cases, even after it has once produced clinical symptoms. Even when dysuria and hemorrhage have set in, spontaneous cure is possible. A sudden renal hemorrhage in a young man, with or without dysuria, and with a more or less regular though slight afternoon rise of temperature, is very likely to be a renal tuberculosis. It is often quite difficult to isolate the tubercle bacilli from the urine in these very early cases, and not a few of the patients go on to spontaneous recovery, that is, the symptoms clear up and the patient has no further trouble.

I have recently seen such a man who has been in perfect health for a long time, although in his early twenties, while engaged in his medical school work, he suddenly developed a renal hemorrhage without the slightest warning and continued to pass blood in his urine for a number of weeks subsequently. He had no dysuria before the hemorrhage set in, but had some while it lasted. I have set him down as a case of cured renal tuberculosis.

On the other hand, one must remember that not all the patients who, after a prolonged period of illness, have their urinary symptoms clear up are really cases of healed tuberculosis. The healing may be apparent, but not real; that is, the ureter may become plugged so that there is no further discharge of pus and bacilli through it into the bladder. Therefore, the bladder symptoms clear up and the tuberculosis is henceforward a local disease in the loin, a tuberculous abscess or tuberculous pyonephrosis, which may undergo further local development or may finally result in systemic infection, or in a local mixed infection with all its disastrous consequences.

When the renal lesion develops so far that the bacilli are readily demonstrable in the urine, then it is time for operation. Such a kidney rarely undergoes a spontaneous cure. It must be excised to prevent the further spread of the disease along the urinary tract, to the opposite kidney and systemically. Tuberculosis of the genito-urinary organs is probably always a secondary lesion, and never primary. Therefore, one always expects to get a well-marked positive reaction with the Koch old tuberculin injection, because of the presence of tuberculous foci elsewhere which flare up in a tuberculin reaction, together with the genito-urinary focus. . . .

Dr. Murphy (February 11, 1915): On further study, chiefly with paraffin sections, this tumor has turned out to be even more interesting in its pathology than we first thought, and justifies the suspicions we had concerning the irregularity in the tuberculin reaction and the misgivings we had concerning the location of the nodule in the upper pole of the testicle. The pathologist reported it originally, after the frozen section examination, as probably a round-cell sarcoma. Our further examination appears to have shown that it is one of those interesting mixed tumors with which Wilms, of Heidelberg, has made us recently more familiar—the embryomata or teratoid tumors. Whereas a true teratoma tends to represent more or less a small fetus, an embryoma represents the three embryonic layers more or less completely, but shows no tendency to form so highly organized a growth as a teratoma. The tissues in the three germ layers of an embryoma each continue to grow like a more or less separate tumor, which may be malignant or non-malignant, but is rather more frequently the former; and when malignant, the different layers may grow through each other in indistinguishable chaos, as is the case here, the cells of the three different germ layers being indiscriminately intermixed. These are the so-called "mixed tumors" of the testicle. Our case appears to be of this chaotic malignant cell type.

Sometimes in these embryomata one sees the cells of a single germ layer predominating in growth over the cells of the two other layers; and then one may have formed an epithelial tumor, an adenoma, for instance, or a cartilage tumor, or a muscle tumor; and scattered among the cells of this predominating cell layer the cells of the other layers will be found in lesser numbers. Sometimes the cells of *two* layers may proliferate to the almost complete exclusion of the third germ layer, making the so-called bidermoma of Wilms.

While these tumors are of great theoretic interest to the pathologist, because of their bearing on the etiology of tumors in general, a subject which Ribbert has taken up nicely in his textbook, they are not of great practical interest to the surgeon because of their rarity. The surgeon's problem is to excise them like any malignant tumor, and that is what we are doing now. We recommended to the patient the excision of both testicles, because we know how apt sarcoma is to recur in the opposite testicle after the excision of the primary seat of the disease; and we fear that an embryoma will follow this general rule. We are not certain on this point, however, and, therefore, the patient has decided to have only the affected testicle and epididymis excised, together with the spermatic cord as far up as we can draw it through the external inguinal tract.

[Dr. Murphy excised the testicle, epididymis, and cord, as described in the CLINICS for February, 1914, page 73, and inserted a paraffin prosthesis as a substitute for the removed testis.] Our experience with this paraffin prosthesis has been very favorable. We insert it rather higher up than we expect it to remain, because its natural tendency is to gravitate a little downward. So far we have always had them heal in by primary intention.

[Note.—The case healed by primary union and the patient has remained perfectly well. A careful examination of the abdomen, both preceding and following the operation, revealed no involvement in the retroperitoneal glands in the neighborhood of the vessels at the hilum of the left kidney. This is a common site for metastatic nodes in sarcoma, as well as carcinoma of the testicle, and examination for these secondary neoplasms should never be overlooked.—Ed.]

TUBERCULOSIS OF THE LEFT SPERMATIC CORD AND EPIDIDYMIS

HISTORY

THE patient, a minister aged forty-one years, entered the hospital April 10, 1915. He has had the usual diseases of childhood; typhoid thirteen years ago (in 1902), and denies venereal infection. His family history is negative to tuberculosis and carcinoma.

August 25, 1914, the patient detected a small nodule, about the size of a hazelnut, just below the external inguinal ring, in the left groin, lying alongside the spermatic cord. This nodule has gradually increased in size since then; soon afterward another nodule developed lower down on the cord, near the upper pole of the testicle, and it, too, has continued to increase in size since first noticed (note the apparently descending course of the infection). The latter is now the size of a filbert. Occasionally, after a day's preaching, he has an aching sensation in the left groin, sometimes radiating down into the testicle. He has had no increased frequency of urination or burning sensation on passing urine, and he has noticed no blood or pus in the urine. Ten years ago he suffered repeatedly with a dull aching in the right lumbar region. These pains often traveled down into the groin and the right testicle, and were especially apt to come on when the patient worked hard. He noticed them every two weeks or so, and they were especially severe on Sundays, after preaching. This complaint persisted until 1913 and then disappeared. He has had no abnormal urinary symptoms at any time, such as burning or increased frequency of micturition, or voiding of blood or pus. For the last year he has urinated once every night, on an average, but believes this due to the fact that he drinks a good deal of water usually before going to bed. He is often restless at night, but has had no night-sweats. He has lost no weight. His bowels generally move twice a day. His appetite is good, and he feels well. He would not have consulted a physician if he had not noticed the nodule in the left spermatic cord.

After entering the hospital he was given a Koch's old tuberculin test, which turned out positive, the highest temperature being 102° F. four hours after the injection. With the fever the patient experienced some general malaise, headache, and a "grippy" feeling in his antrum and through the back of his head.

Examination by Dr. Murphy showed a tumor, apparently, of the vas, just above and in front of the upper pole of the left testicle, and a second nodule at the external inguinal ring, also on the vas. From the irregular induration of the cord between the two nodules and the positive tuberculin test it was decided that an operation was necessary for the removal of these probably tuberculous lesions. The cord at its lower end appeared to run to the front of the testicle, instead of to its posterior surface, as normally, probably because the lower nodule on the vas is adherent to the anterior surface of the testicle. It is scarcely possible that a torsion of the spermatic cord nearly 180 degrees in extent can have taken place in the entire absence of its usual symptoms in the history, particularly pain, which is an almost invariable accompaniment of a twisted cord.

COMMENTS AND OPERATION

Dr. Murphy (April 12, 1914): What symptom did he notice first?

Intern: A nodule about the size of a hazelnut just below the left external ring, apparently lying in the spermatic cord.

Dr. Murphy: That is a finding to which I wish to call your attention particularly, because it is a very exceptional occurrence if the condition present here is a tuberculosis. Note that the disease according to the patient's history, began as a nodule located just below the external inguinal ring. That location is extremely rarely the primary seat of tuberculosis; but this patient is an educated and intelligent man, and describes the course of his condition with accuracy of detail, and I am always greatly disinclined to question the truth of such a patient's statements. There is still another point in the history which is out of tune with the diagnosis of tuberculosis of the epididymis or vas. One of the first symptoms of genito-urinary tuberculosis, whether primary in the kidney or in the epididymis, is increased frequency of urination. This, we believe, is due to the irritation from the discharge into the spermatic vesicles and bladder of material from the tuberculous foci. This patient tells us nothing of any such complaint; in fact, he denies it strenuously. This is quite contrary to the rule. Usually these symptoms are what the patient comes into the hospital complaining of. Increased frequency of urination is the most constant of all the early symptoms or signs of genito-urinary tuberculosis. It often precedes the presence of an increase in the number of urinary leukocytes, and, in a very large percentage of the cases, occurs before it is possible to demonstrate tubercle bacilli in the urine by staining or by inoculation of guinea-pigs. Perhaps this patient urinates a little more frequently than normal but does not realize it. At least we know, from his own statement, that he rises every night to urinate.

There is another early symptom often seen with tuberculosis of which the history tells us nothing. When these patients have increased frequency of urination, the demand to urinate becomes very urgent with them, and the patient must run to the toilet immediately. This patient's subjective symptoms, you see, were not sufficiently marked to bring him to a doctor. It was the evident physical abnormality in the cord which made him seek medical advice. He is a powerful, athletic man, as you see, not a likely looking subject for tuberculosis, and he has no history of trauma to this region; and a history of trauma, you know, is a frequent precursor of tuberculous lesions here as well as elsewhere. What about the tuberculin test?

INTERN: It was positive.

DR. MURPHY: The patient complained of a "grippy" feeling in his antrum, general malaise, and headache after the tuberculin injection. He has had some antrum inflammation for years, and the injection seemed to stir up his trouble somewhat. His temperature rose to 102° F. That is a definitely positive reaction, and since the patient has no evidence of tuberculosis elsewhere, we have good reason to presume that these lesions in his epididymis and cord are tuberculous.

[The usual oblique inguinal incision was made.]

There is the inguinal nodule, you see, and here is the second mass at the upper pole of the testicle, where the globus major of the epididymis is fused with the upper pole of the testicle in front. It looks, indeed, as if there has been some torsion of the testicle. Remember that the epididymis and vas normally lie on the outer and posterior surface of the upper pole of the testicle. Note, too, that the vas here lies on the outer surface of the cord; evidently the cord as well as the testicle has been twisted on its long axis. This torsion may have been due to the disease present or—it is quite as likely—the disease may have been due, in large measure, at least, to the torsion. The vas is very extensively involved in the disease, which appears to extend in the cord, even somewhat above the nodule at the external inguinal ring. I am now fairly certain that the condition is a tuberculosis, although old inflammatory adhesions are so numerous here that it is difficult to note any individual isolated tubercles.

Let the record show that a torsion of the testicle has taken place here about its long axis and is nearly 180 degrees in extent. We must, therefore, remove the testicle. We cannot allow a pathologic organ of this type, with so highly developed a twist in the cord, to remain when other severe pathologic changes are also present.

In analyzing this case we considered the possibility of a sarcoma of the testicle with metastasis in the cord at the external ring; but we rather concluded, on the basis of the positive tuberculin reaction and the fact that the mass at the upper pole of the testicle was inseparable from the cord and epididymis, that we probably had to deal with a tuberculosis primary in the globus major of the epididymis, and extending upward into the vas and cord, although the reverse order of spread had been apparently observed by the patient. But primary tuberculosis of the spermatic cord or vas is a very rare lesion, and so is tuberculosis of the body of the testicle (*rete testis*), which in the few cases in which it does occur is usually found at the hilum or near the hilum of the gland. The history and the objective findings clashed in this case, but in such conditions one must cling to what he observes himself, rather than to what he hears from the patient, however sincere the patient's statement may seem to be.

Let the record show that there is no evidence of disease in the opposite testicle.

After operating on the scrotum we always wipe out the wound with 5 per cent. carbolic acid solution, because the hands touch the scrotum during the operation, and therefore are liable to carry infection into the wound.

Let the record show that the left testicle, epididymis, and spermatic cord were excised. The cord was pulled down as far as possible and then ligated and cut, the nodule at the external ring coming away with it. The vas was isolated, and the mucosa inside the stump was cauterized with 95 per cent. carbolic acid on a toothpick to prevent a tuberculous fistula subsequently, which is liable to develop if the infected mucous membrane of the vas is left untreated.

This was probably one of those cases of congenital torsion of the testicle. When I first examined the patient I noticed that the cord ran to the anterior surface of the testicle and ended there in a nodule. I did not immediately associate this finding definitely with a torsion of the cord, because of the entire absence of symptoms, such as one always finds with an acute torsion. It is very rare that a torsion of the cord nearly 180 degrees in extent does not produce active symptoms. A turn of 90 degrees usually produces venous blocking, and a turn of 180 degrees arterial blocking also and complete gangrene of the testicle. In the absence of any history of active symptoms I must conclude that this torsion was congenital in origin. That is probably the reason why the testicle is not necrotic or even atrophic; but it may also be the reason why tuberculosis attacked this organ in an otherwise healthy man. It is often of vital importance to recognize the presence of testicular torsion, especially when the use of the trocar or scalpel is under consideration. In this case, while we suspected its existence, it was not a matter of vital importance to recognize it definitely, which we could have done, I think, had we had any reason to suspect its presence from the symptoms or the history of the patient. There is nothing in the patient's statements that gives us any clue on which to base a suspicion that this is an acquired and not a congenital lesion.

Visiting Doctor: If there had not been extensive pathologic changes present, what would you have done with the twisted testicle?

Dr. Murphy: I should have untwisted and slightly overcorrected it, and then fastened it with a stitch at the bottom of the scrotum. That procedure pretty effectively prevents recurrence of the torsion.

Visiting Doctor: When the testicle is involved in the spread of tuberculosis from the epididymis, do you resect part of it, or do you excise the entire testicle?

Dr. Murphy: That will depend largely on the extent of the disease. If the involvement of the testicle is not very extensive, I usually excise the lesion by resecting that part of the testicle containing it; but when the testicle is extensively involved, I think it is best to remove the entire gland. We know that after partial excision a considerable amount of atrophy of the testicle ensues; but experimental work shows that, however atrophic the sperm-producing parenchyma becomes, the interstitial cells on which the internal secretion of the testicle probably depends persist, and presumably functionate. To leave in a small portion of a badly diseased gland is a purely sentimental procedure, adds little or nothing to the sum total of internal secretions, and invites fistula formation and subsequent infection of the opposite testicle. We can later insert a paraffin prosthesis in this side of the scrotum to replace the testicle if we get primary healing.

[Pathologist's Report: The excised specimen, consisting of testis, epididymis, and spermatic cord, showed a well-advanced fibrous and caseous tuberculosis.]

[Note.—Primary union; tuberculin treatment was ordered, and instructions given that it be continued for at least six months. —Ed.]

CHRONIC TENDOVAGINITIS OF THE EXTENSOR TENDON OF THE THUMB.—EXCISION OF THE TENDON-SHEATH WITH THE UNDERLYING PERIOSTEUM.—NEW TENDON-SHEATH MADE FROM PEDICLED FLAPS OF FAT

HISTORY

THE patient is a farmer, forty-one years old. He entered the hospital October 3, 1914. He has never been ill previously except for the usual diseases of childhood and an attack of typhoid fever in 1898. His family history is negative as to both tuberculosis and malignant tumors.

The patient's present trouble began in March, 1914. He was trimming a horse's hoof when the animal became frightened, jerked away, and twisted the patient's left hand and wrist, paining the latter considerably. Following this accident a small swelling developed on the radial side of the left wrist, just proximal to the joint. It grew to about the size of a split walnut in the month following the accident. It felt hard and was tender on deep palpation. It usually seemed to increase somewhat in size after a day's hard work. The patient can push or pull in a straight line without producing pain in the wrist; but movement of the wrist from side to side, that is, ulnar flexion and radial flexion, is always painful. He can flex and extend the hand and all the fingers without difficulty or discomfort. He has never had any chills or fever, to his knowledge, nor any night-sweats.

COMMENTS AND OPERATION

DR. MURPHY (October 3, 1914): This man has but little to show in the way of deformity, and yet he is practically incapacitated for his usual work because of this lesion of the left wrist.

When he first came to us we feared that he was developing a sarcoma of the lower end of his radius. A growth at the lower end of the radius, developing gradually and relatively painlessly following a twisting or wrenching trauma is very likely to be a bone sarcoma. Remember, this was only a moderate trauma which the patient suffered, and it is after the moderate traumas, especially those of a twisting or jerking nature, that sarcoma is prone to develop. Severe traumas, such as produce fracture or dislocation, are very rarely, if ever, followed by the development of sarcoma. We also thought of the possibility of a periostitis, either traumatic or metastatic, developing in a *locus minoris resistentiæ* produced by the injury. Such a metastatic periostitis often follows an acute tonsillitis, an acute pharyngitis or rhinitis, typhoid fever, and like infections. The x-ray plates, however, eliminate these possibilities at once. (See the accompanying roentgenograms.) The bones of the right wrist appear perfectly normal. There is no tumor within the radius or springing from its periosteum, and there is none of the ossifying periostitis present which is always to be seen in the x-ray plate of a metastatic periostitis of this duration. We can completely rule out a chronic periostitis. There are no arthritic changes visible in the wrist-joint and carpus in the x-ray plate; but we could easily rule out the possibility of this swelling being due to a chronic inflammation of the wrist-joint by the clinical manifestations, even if we did not have the x-ray plate at hand.

Therefore, only two possibilities appear to remain—the development of an adventitious bursa over the prominence of the radial styloid, quite a rare occurrence, or the development of a ganglion, a chronic tenosynovitis, of the extensor tendon of the thumb, one of the commoner seats of ganglion formation. The extensor tendons of the thumb seem to run right over this prominence, and movements of the thumb, especially flexion, are quite painful. The tumor feels hard, not fluctuating; but remember that encysted fluid under tension always feels like a hard tumor if the tension be great.

[Dr. Murphy made a longitudinal incision over the apex of the swelling, as shown in the accompanying drawing. By dissec-

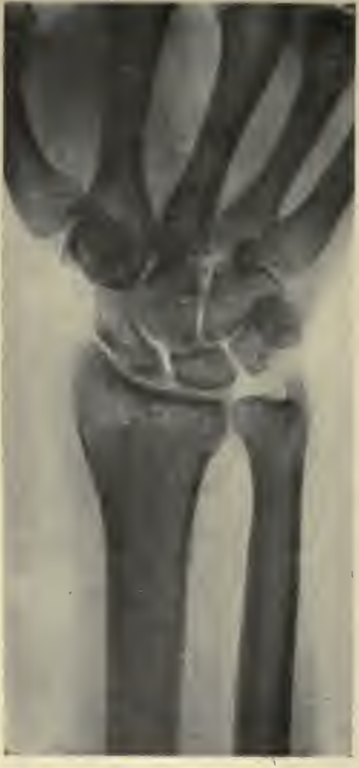


Fig. 135.—Tendovaginitis of extensor tendon of left thumb. The *x*-ray plates show the bones of the left wrist perfectly normal, thus eliminating the possibility of sarcoma, periostitis, or osteomyelitis as the cause of the swelling located over the styloid process of the radius. Anteroposterior roentgenogram before operation.

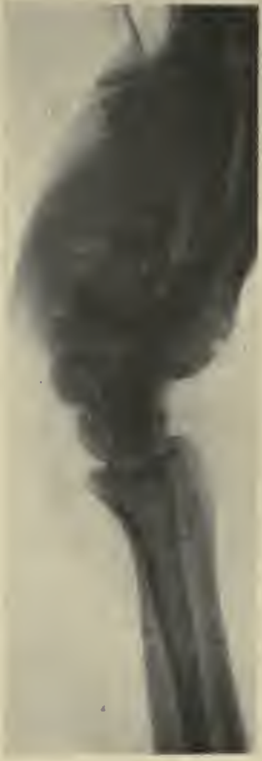


Fig. 136.—Tendovaginitis of extensor tendon of left thumb. The *x*-ray plates show the bones of the left wrist perfectly normal, thus eliminating the possibility of sarcoma, periostitis, or osteomyelitis as the cause of the swelling located over the styloid process of the radius. Lateral roentgenogram before operation.

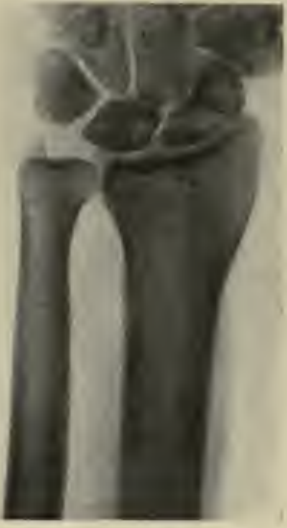


Fig. 137.—Tendovaginitis of extensor tendon of left thumb. This anteroposterior roentgenogram of the normal *right* wrist is reproduced for comparison with the also normal appearing left wrist, to show that the swelling noted just above the left wrist lies entirely in the soft parts and not in the bone.

tion he gradually outlined the growth and found it confined to the sheath of the extensor tendon of the thumb.]

This is evidently a tendovaginitis of the extensor tendon of the thumb. The lower surface of the sheath is firmly adherent to the periosteum of the radius. I will open the ganglion and see what its contents look like. . . . See the fluid under tension! See how it spurted out as I punctured the sac! The fluid appears perfectly clear and contains no foreign material, like rice bodies. Now I have slit up the sac longitudinally, and as I explore it carefully I see no thickenings or deposits like tubercles on its walls. There seem to be a few flakes like fibrin in the contained fluid.

What shall we do next? We have taken the tension off the swelling by letting out the fluid, but if we were to sew up the cyst-wall again, the fluid would reaccumulate, in all probability. The only sure method of avoiding a recurrence is to excise the sac completely, and that is what we are doing. (See the accompanying drawing.) The posterior surface of the encysted sheath seems even more thickened than its anterior surface, and it is firmly adherent to the periosteum of the bone beneath. The adhesions are so firm and dense that I presume they were inflammatory in origin. Whether a traumatic periostitis involved the tendon-sheath secondarily, or whether a traumatic tenosynovitis underwent secondary infection with germs circulating in the blood and thus led to a superficial periostitis by direct extension through the sheath, I do not know. The result is the same. I must dissect the involved periosteum from the bone, together with the tendon-sheath to which it is adherent.

The excision is complete, but the raw surface of the bone is now left exposed. How shall we cover it? How shall we keep the extensor tendon from adhering subsequently to this raw surface and becoming as greatly limited in its movements as previously? This question is easily answered. We shall simply prepare two pedicled flaps from the fat on either side of the denuded area, and draw these flaps together beneath the tendon and over the bone, thus separating the two structures perfectly and permanently, and giving the tendon a smooth

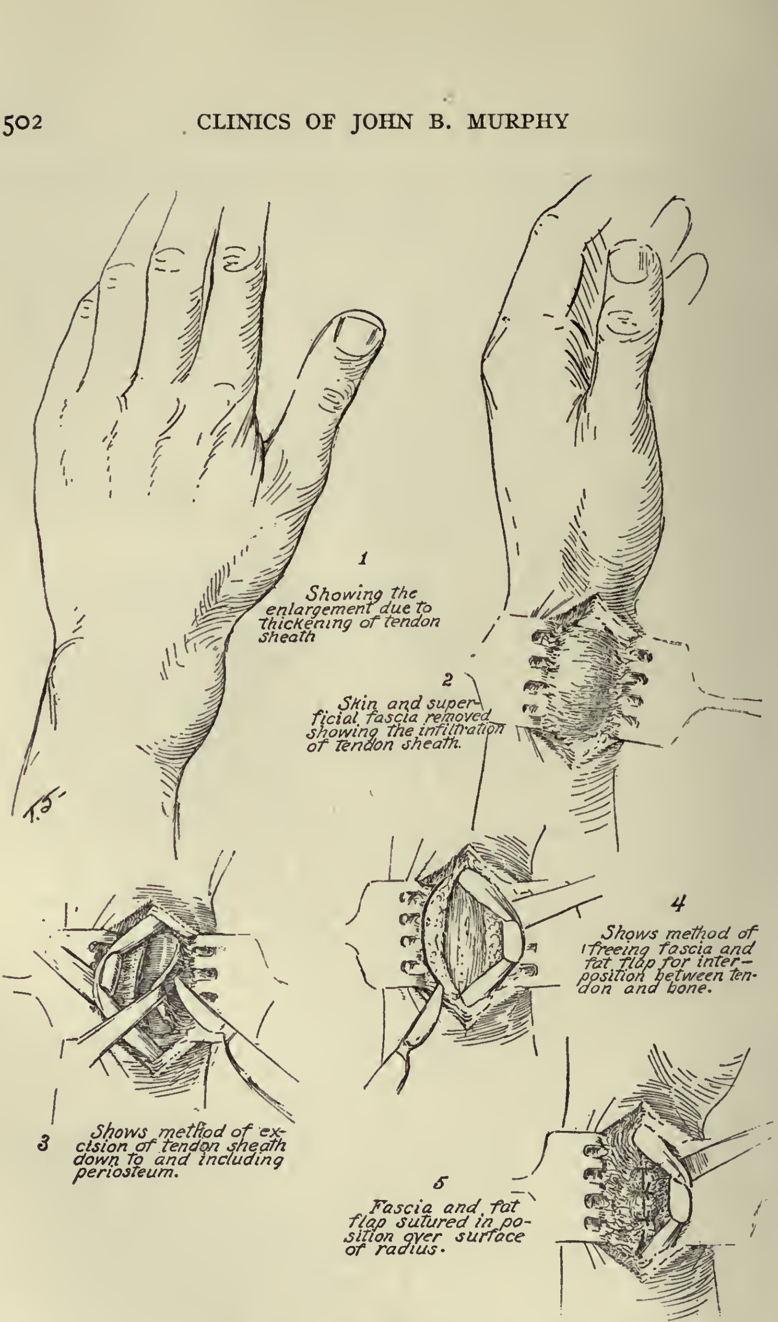

1
Showing the enlargement due to thickening of tendon sheath
2
Skin and superficial fascia removed showing the infiltration of tendon sheath.
3
Shows method of excision of tendon sheath down to and including periosteum.
4
Shows method of freeing fascia and fat flap for interposition between tendon and bone.
5
Fascia and fat flap sutured in position over surface of radius.

Fig. 138.—Tenosynovitis of left wrist (sheath of extensor pollicis involved). Excision of chronically inflamed sac and underlying periosteum. Formation of a new bed for the tendon by turning a pedicled flap of subcutaneous fat and fascia over the denuded bone, between it and the tendon.

surface to move over. (See the accompanying Fig. 138.) The tendon will also be covered by fat on its upper surface, that is, by the subcutaneous fat. Therefore, being surrounded completely by fat, its movements will not be interfered with by the subsequent development of scar tissue, and a fatty bursa will probably develop around it in the course of time, as is usually the case. The determining factor in the development of such a postoperative bursa appears to be the amount of subsequent use of the thumb. If the patient continues to manipulate and passively work his thumb immediately after operation and subsequently, he will get a good result; but if such a thumb is immobilized for a period of weeks after operation, connective-tissue adhesions are much more likely to develop than is an adventitious bursa. I think that something like this same process occurs following my arthroplasties. At any rate, those patients who follow the after-treatment thoroughly and painstakingly get good results. Those patients who complain persistently of the slight discomfort which passive movements cause after operation do not get such good results. For a good cosmetic and functional result women seem to be more ready to pay the price than men, and for my arthroplasty patients I prefer the former. I presume that I am not the first to discover that women, at least some women, are willing to value appearances above comfort. Some men are, too, but not so many, at least in my experience.

PAINFUL EXOSTOSIS OF THE OS CALCIS

HISTORY

The patient, a single man, aged twenty-one years, entered the hospital September 30, 1914. His family history is negative as to tuberculosis. In November, 1913, he contracted a "chest cold," was in bed for one week, and had a fever which ran as high as 102° F. He coughed but little while in bed, but after getting up he coughed considerably during the ensuing four days, and expectorated profusely. The sputum was thick and yellow, but showed no blood. (Apparently an acute bronchitis.) Since then he has had no cough worth mentioning and no fever. On three different occasions, however, he has taken tuberculin for several weeks at a time, on the advice of his physician. He has always felt well after taking it and has gained in weight.

His present trouble dates back to three years ago (1911). The patient then began gradually to develop pain in the region of the left heel, more on its plantar surface than posteriorly. The pain seemed more marked in the morning just after rising. After the patient had taken a few steps about the room the pain usually diminished until it became barely perceptible. From month to month, however, the annoyance gradually increased. Six months after the pain was first noticed it had become especially bothersome on long standing and following much walking or running.

In July, 1912, the patient jumped from a slowly moving wagon, landing on the ground on both heels. This fall caused him such severe pain that he was unable to walk because of it, and had to be carried home. On the following day, however, he felt much better and was up and about again. He noticed, not long after this time, that his left tendo Achillis was constantly not only tender, but also somewhat swollen. His right heel gradually became similarly swollen and also very painful.

He consulted several physicians. They differed as to both the diagnosis of his ailment and the proper treatment for it. These medical disagreements were confusing to him, and made him doubt the doctors' knowledge. Some stated he had rheumatism; others said bursitis; some advised heat; some cold; some the rest cure, and some exercise. He followed a little of the advice, but felt no better. Then he took to wearing shoes with a round, shallow depression cut out on the inside of the heel, in order to make more room for the heel; but he experienced very little relief.

So long as the patient is in bed he suffers no pain, but his feet are tender to pressure, particularly deep pressure. There is no visible swelling of the heels at present, and no history of chills, fever, night-sweats, or coughs, with the exception of the "chest cold," mentioned above. In February, 1913, a Wassermann test was made and was reported negative. The patient himself denies positively venereal infection. He gives no history of trauma preceding the onset of his pain.

COMMENTS AND OPERATION

Dr. Murphy (October 13, 1914): What relation did the "chest cold" have to the development of the pain in his heel? When was this cold?

Intern: In November, 1913.

Dr. Murphy: Then it had no relation, of course, to the onset of his trouble. You said something about the back of the heel being swollen.

Intern: The tendo Achillis was somewhat swollen laterally.

Dr. Murphy: What do you mean by laterally?

Intern: On the side of the heel.

Dr. Murphy: I think you mean that there was some swelling present on both sides of the tendo Achillis, obliterating the normal depressions there. Is that all your history?

Intern: That is all.

Dr. Murphy: I will go on with this from where the history

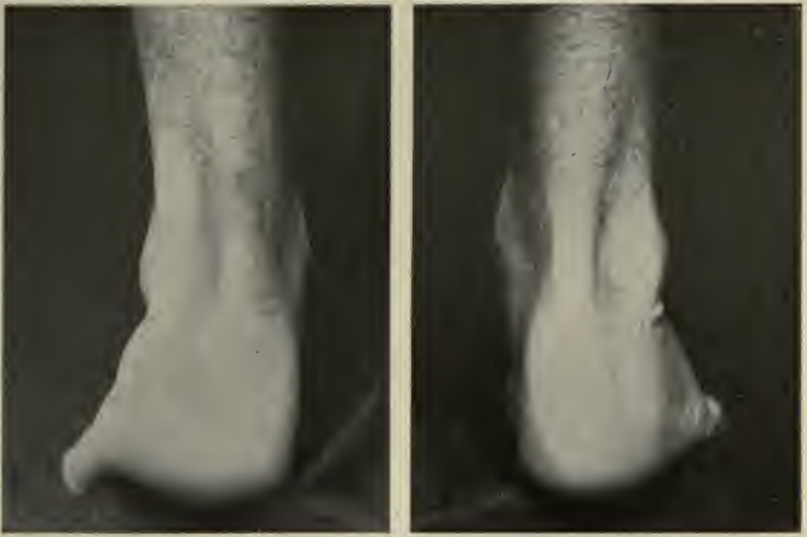


Fig. 139.—Multiple painful exostoses of the os calcis with accompanying bursitis. Note the fullness of both heels, especially of the left, over the upper third of the tuber calcanei, owing to the distention of the sub-Achilles bursa with fluid, the accumulation being due to the accompanying chronic bursitis.

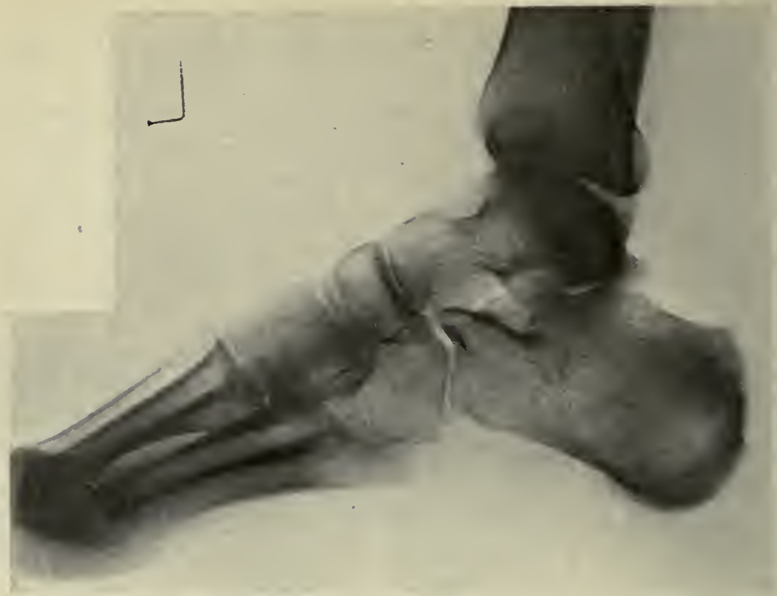


Fig. 140.—Multiple painful exostoses of the left os calcis, with accompanying bursitis. The cartilaginous exostoses do not show in the *x*-ray plate; only the osseous exostoses, and the latter are relatively insignificant in size.

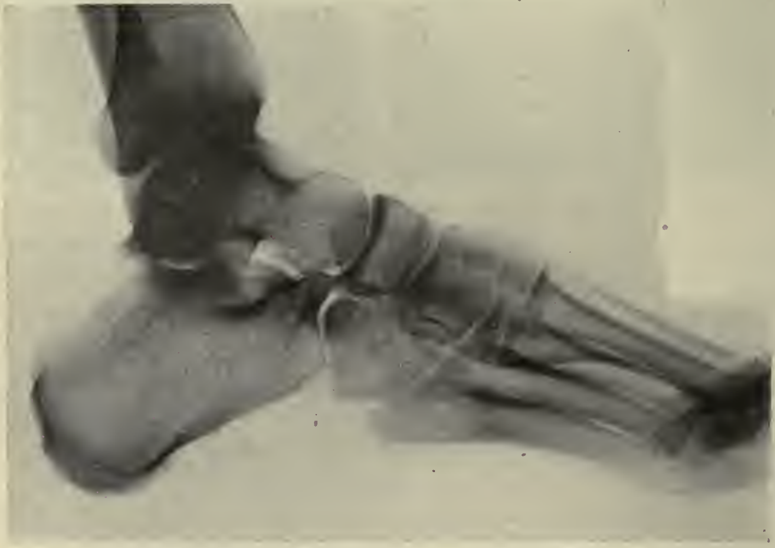


Fig. 141.—Multiple painful exostoses of the os calcis with accompanying bursitis. Lateral roentgenogram of the right foot. The plantar calcaneal exostosis is the only one to be seen on this *x*-ray plate. The affection was only slightly developed in the right foot as compared with the left, and the bursitis predominated over the "spur" formation.

stops. It is a very instructive and rather exceptional case. The *x*-ray plate goes a long way toward making the diagnosis. The patient's pathologic condition is a low-grade inflammation of the little bursal pouch which lies between his right calcaneus and the tendo Achillis, the so-called bursa Achillea anterior or retrocalcanea. At least that is his principal complaint.

In this lateral *x*-ray plate [demonstrating] one does not see the bursitis, of course, but one sees the results of its action on the bone—first, erosion of the bone, and, second, osteophyte production as an expression of the irritation of the surrounding periosteum. One of these inflammatory exostoses lies just at the attachment of the tendo Achillis, as is so frequently the case. Inflammatory osteophytes are very likely to localize at tendon insertions. There is another exostosis on the plantar surface of the calcaneus, at the point of attachment of the plantar ligament, and the light irregularity of the surrounding bone rather suggests that there is a bursitis here, although it is the Achilles bursitis which is causing him the most pain. This predominance of the posterior pain is rather contrary to the rule in such cases, for it is the plantar pain, usually, which predominates.

The *x*-ray picture of the right foot appears approximately normal. Although at times it has caused the patient some annoyance, the bursitis of the right heel has not yet developed sufficiently to produce bone changes of a noticeable degree.

Such a bursitis usually produces a distinct difficulty in gait, which may be rather characteristic of the affection. When this young man walked into my office he came in on his toes, like this [indicating]. He did not allow his heels to touch the floor, because of the pain produced.

Why he has pain on touching his left heel to the ground is quite clear when one looks at the *x*-ray plate. When the patient rises on his toes the Achilles tendon is considerably separated from the os calcis; but when his sole is flat on the floor the Achilles tendon is closely approximated to the calcaneus and compresses the inflamed bursa between it and the bone.

Most of the patients who complain of pain like this in the heel are ordered to put on low-heeled shoes, there being a general impression among members of the profession, as well as among the laity, that they are more "hygienic." Low-heeled shoes are just what these patients should not wear. Not only do they cause pain and great discomfort, but they actually aggravate the disease by putting the products of inflammation under pressure. What should be done is to elevate the heels of the shoes, so as to take tension off the Achilles bursa by increasing the angle between the tendon and the bone, and lessening the tension of the tendon itself. When the pain is located on the under surface of the heel and is caused by inflammation of the bursa over a plantar exostosis of the calcaneus, high heels are again much preferable to low heels, for they take tension off the plantar fascia, which compresses the plantar bursa, just as the Achilles tendon compresses the retrocalcaneal bursa. With elevation of the heel discomfort is greatly diminished in both instances and may entirely disappear. In recommending high-heeled shoes, I am recommending them, of course, only for these patients, and not for healthy individuals, for whom, probably, low-heeled shoes are more "hygienic" and less likely to produce such annoying defects as corns and bunions.

This boy is practically out of commission as a useful citizen because of the intolerable pain which the bursitis causes him. He has been long unable to do any work requiring his standing for a considerable period of time. The doctor, in his history, did not speak of a complication in the case which should be mentioned. Ever since the boy's "chest cold" in 1913 he has had something of a cough, and we considered the possibility of this bursitis being tuberculous in nature. We, therefore, made the tuberculin test. He gave a positive general reaction with the first test, his temperature rising to 103° F. There was, however, no local reaction in either foot. There was no swelling and there was no increase in sensitiveness. Therefore, we rather suspected that a small tuberculous focus might lie back of his cough, although physical examination was negative and the *x*-ray plates of the chest showed no definite pathologic change. His sputum was repeatedly examined for tubercle bacilli, and none were ever found. We then put him on tuberculin treatment, and his cough has materially diminished—practically disappeared. He is still in bed, because his feet are much too painful to get about on, and probably this rest in bed, full feeding, and tuberculin therapy acted together to relieve his pulmonary lesion.

Not only does he have the intense pain on walking, but direct pressure over the bursa is as painful as pressure over a fresh furuncle. If you press on the bursa unexpectedly, he actually screams with pain. To rid him of his trouble we must remove not only the calcaneal spur, but also the entire bursa and the surrounding periosteum; that is, the trouble must be excised completely as well as the tissues which have been subjected to this chronic pressure and to the action of the inflammatory products. If we do not excise the periosteum, it will be likely to remain painful, and possibly produce more painful osteophytes later.

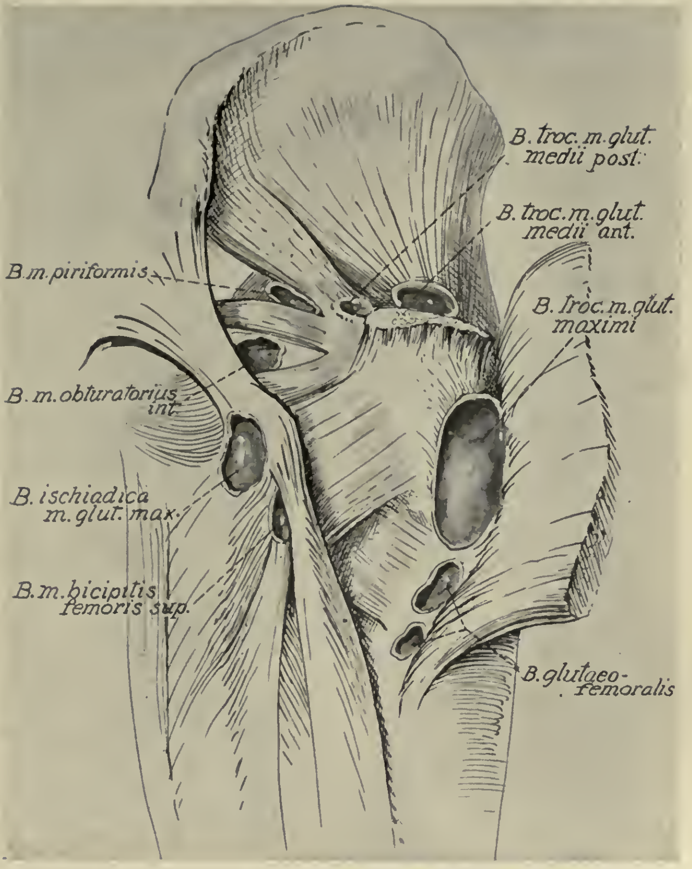
Pain just like this is seen on pressure over bursitides elsewhere, such as the not infrequent bursitis occurring in the deep bursa over the great trochanter. Have you ever seen a case of chronic bursitis in the trochanter region? If you have had one to treat you will probably remember it as long as you live. At first there seems to be only a small quantity of fluid just over the bony prominence. The patient complains of occasional or frequent pain over the bursa when the fascia is put on the stretch, and the spot is extremely sensitive to pressure. It looks like a small undertaking, so you operate and remove it. Very likely you find rice bodies in it, and after you have completed your dissection you tell the patient he is cured; but he is not cured—in all likelihood he is just beginning. The affection comes back in two months or so. You operate again, making up your mind that you must have left a portion of the bursa before. You tell the patient, "Well, this time I got it all." But again it comes back. Usually you operate until you get some little experience with this particular lesion. Finally, in desperation, you start your incision somewhere near the anterior iliac spine and dissect down to somewhere near the external condyle of the femur, removing everything in sight that looks like a bursa or looks as if it might develop into one; and thus at last you extirpate the disease. A case like that will thoroughly convince one of the great natural tendency of a synovial cavity to form where conditions are favorable for it, and to confirm one's faith in the great future of plastic work on joints.

[That Dr. Murphy's method of arthroplasty, using a fascia and fat flap for interposition between the bones, is becoming the standard technic, not only on this side of the water, but also abroad, may be seen from the fact that Professor Payr, who originated and successfully used the method of free transplantation of joints, has given up his plan in favor of Dr. Murphy's procedure. Dr. Payr has recently published in the *Münchener medicinische Wochenschrift* for January 26, 1915, two cases of knee arthroplasty performed by Dr. Murphy's method. The results were knees anatomically and functionally practically perfect, results as good as Dr. Murphy has obtained in many of his cases. Since success can thus be attained by any first-class surgeon, as it has been attained both here and abroad, it would seem that this operation is bound to become a standard practice. An abstract of Professor Payr's article appears in the *Journal of the American Medical Association* for March 13, 1915, page 950:

"*Outcome of Operative Mobilized Joints.*—Payr reports with pride that two cavalry officers have gone through the campaign to date without the least disturbance from the knee which he had previously operated on. He had mobilized the totally stiff joint and interposed a pedicled pad of fascia and fat. This has restored practically normal function to the joint, even through the stress of the active campaign, which he regards as the severest test of an arthroplasty that can be imagined."]

VISITING DOCTOR: Did I understand you to say that the trochanteric bursa may extend from the superior iliac spine to the knee?

DR. MURPHY: I am speaking, of course, of a pathologic bursa, and not of the normal bursa. A bursa tends to spread and burrow through the tissues so long as it is chronically infected and contains fluid under tension. Bursæ are not stable anatomic structures. Only the larger bursæ are constantly found at birth. The smaller bursæ develop wherever there is need of them, par-



B. troc. m. glut. medii post.
B. troc. m. glut. medii ant.
B. m. piriformis
B. troc. m. glut. maximi
B. m. obturatorius int.
B. ischiadica m. glut. max.
B. m. bicipitis femoris sup.
B. glutaeo-femoralis

Fig. 142.—A diagram showing the location and relations of the commoner bursæ in the region of the trochanter and hip. (The B. N. A. nomenclature is used for the sake of uniformity. No distinct names were provided for most of these bursæ in the old nomenclature.)

ticularly where there is pressure over bony points. In a region such as the hip, where there is naturally much pressure, muscular and traumatic, over certain bony points, there are many bursæ, and when one or more start to spread under the influence of chronic inflammation, they may fuse one with another until a long chain may finally unite to form a single continuous endothelial space. These spaces are very likely to communicate with the neighboring joint also, and when excised, the endothelium may at times regenerate even from the endothelium of the associated joint. At any rate, we know that these large chronic inflammatory bursæ in the course of time may become extremely difficult to extirpate entirely.

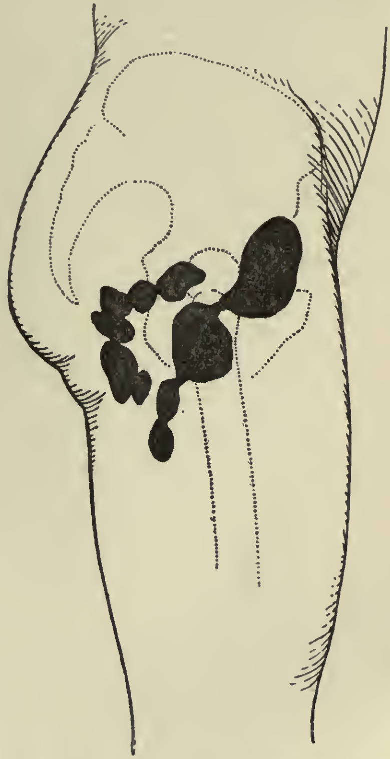


Fig. 143.—Diagram showing how bursæ enlarged as the result of a chronic bursitis fuse with neighboring bursæ to form long connecting chains, often communicating sooner or later with the neighboring joint. Diagram drawn from an injected anatomic preparation of the bursæ around the hip.

Over the trochanter there are two bursæ, one deep and one superficial. The deep bursa is more constantly present than the superficial and gives more trouble, because it is larger, burrows more easily along the fascial planes, and more frequently communicates with the neighboring bursæ, particularly the iliac bursa, which in turn not infrequently communicates with the hip-joint itself. Inflammation in the deep bursa produces a swelling at the upper border of the greater trochanter and along the external surface of the femur. As the swelling increases the gluteal fold may become obliterated above, and the swelling also gradually extends below, beneath the tensor fascia femoris. As the bursa spreads it not infrequently develops a communication with the bursa beneath the gluteus medius. When large, such a bursa may appear to be divided into two portions by the lower tense border of the gluteus maximus running across it.

There is a typical position of the limb in trochanteric bursitis, just as there is a typical position of the foot—plantar extension—in Achilles bursitis. The limb in bursitis, just as in arthritis, is held in that position in which tension on the inclosed fluid is at a minimum. The usual position in trochanteric bursitis is flexion, abduction, and external rotation, the gluteus and tensor fascia femoris being thus somewhat relaxed. Much more rarely one sees adduction and inward rotation.

When one first sees these cases coxitis is usually immediately thought of; but the hip-joint can be moved painlessly, there is no pain on jarring the hip, there is no adductor spasm, and there is tenderness over the bursa, a tenderness which is absent in coxitis. When suppuration takes place in large bursæ, numerous cutaneous fistulæ often develop. Movement is limited by the subsequent development of scar tissue, and the resemblance to hip disease becomes still more striking. Of course, when such an infected bursa communicates with the iliac bursa, and the iliac bursa with the joint, an acute suppurative coxitis develops. It is usually not easy in such cases of extensive suppuration to determine at operation or even at autopsy where the disease was primary.

An acute osteomyelitis of the trochanter is often even more difficult to differentiate from acute suppurative bursitis than is a coxitis, because the secondary periostitis which develops in consequence of the osteomyelitis lies so close to the bursa that it produces practically the same local findings as a bursitis, and may actually involve the bursa secondarily. The general symptoms of trochanteric osteomyelitis, however, are much more severe than those of a bursitis, and the operation which drains the pus focus at the same time reveals the site of the disease in the bone.

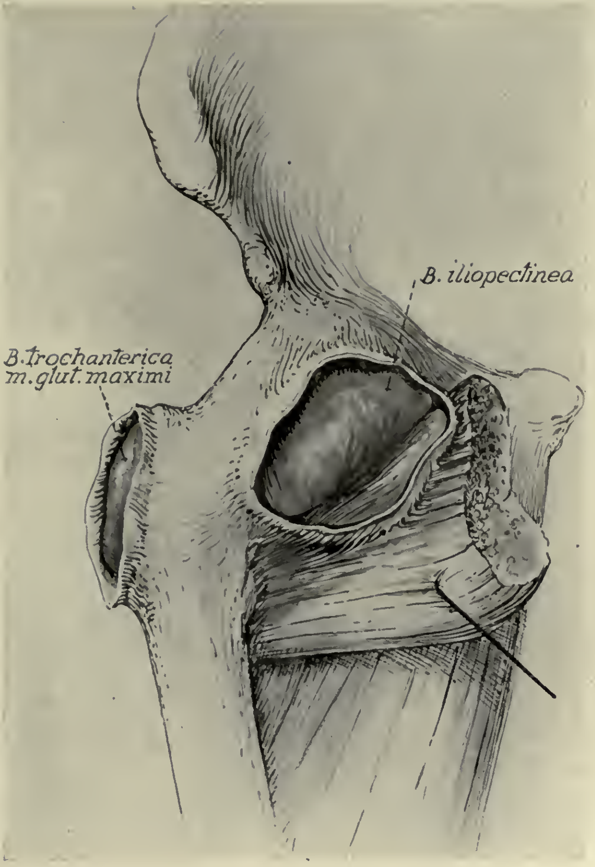


Fig. 144.—A drawing showing the iliopectineal and deep trochanteric bursæ somewhat enlarged as the result of a chronic bursitis.

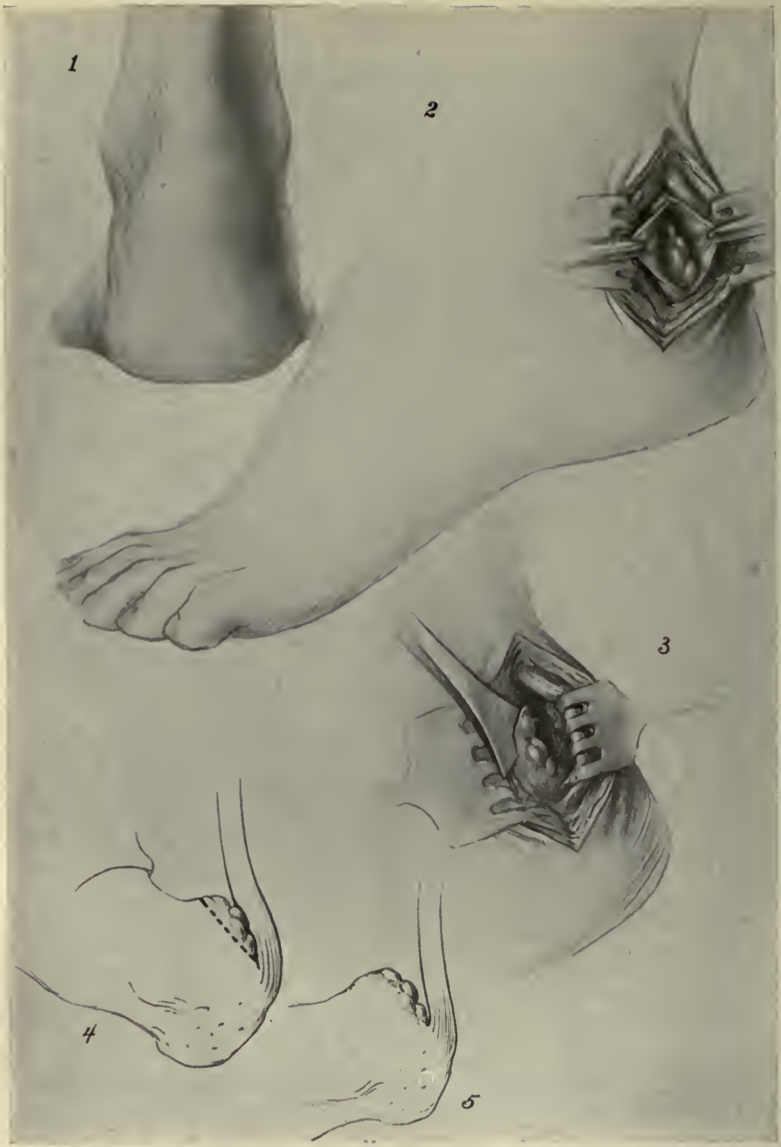


Fig. 145.—Painful exostoses of the os calcis, cartilaginous and osseous, the result of chronic bursitis, probably metastatic in origin.

1, Shows the broadening of the heel over the upper third of the tuber calcanei, due to the accumulation of fluid in the sub-Achilles bursa.

2, Bursa opened, showing the cartilaginous exostoses. Note the generous size of the bursa, much larger than normal, as is the case almost always in chronic bursitis.

3, Showing the method of removing the exostoses, together with the underlying bone and the surrounding area of periosteum, in order to guard against recurrence. Note how well adapted to the purpose is the artist's chisel used. (The steps of the subsequent excision of the bursa are not shown.)

4, Showing how, in dorsal flexion of the foot, as in walking, the exostoses pressed on the under surface of the tendo Achillis and produced great pain. A high-heeled shoe diminishes dorsal flexion and so lessens the pain caused by such a lesion (Murphy's sign). The dotted line indicates the direction of excision of the exostoses.

5, Showing why there is relief from pain when the foot is in plantar flexion.

The disease is further developed in this patient's left Achilles bursa than in the right. The right heel gives less discomfort and shows less change in the *x*-ray plate. My diagnosis is that this is a metastatic bursitis, coming either from some attack of pharyngitis or tonsillitis, or from the patient's chronic bronchitis. I do not believe that the bursitis is tuberculous, because of the complete absence of local reaction following the tuberculin test.

I am making my first incision longitudinally along the inner border of the Achilles tendon. Here the scar will be very little exposed to subsequent trauma. With the first incision I come down to the periosteum. Note how edematous and thickened it is. My knife seems to penetrate it for nearly a third of an inch before it strikes bone. Some of the bursal fluid is now flowing out into our field. There seem to be a number of fat-globules in it; but otherwise it is relatively clear. There are often tags of fat in such bursæ.

I can feel no rice bodies anywhere, but I do find a number of exostoses on the back of the calcaneus. As my assistant retracts the edges of the wound I want every one to get a good look at these. They seem to be chiefly cartilaginous, and that explains why they did not appear in the *x*-ray plate. Cartilage in the *x*-ray casts practically no more of a shadow than does muscle. Only that portion of these exostoses shows in which there was actual calcification; and, therefore, we had no suspicion, from looking at the *x*-ray plate, that we should find so many and such prominent knobs in this location. When one considers that with every step the Achilles tendon rubbed over these inflammatory cartilaginous exostoses, with their sensitive perichondrium, one can readily understand that the patient was out of commission for any work which required the standing position.

Not only do these little pegs catch on the Achilles tendon, but also on the upper rim of the heel of the patient's shoe—the so-called counter. To rid the patient of his trouble we must excise every portion of the bursa, remove all these excrescences, and also the surrounding periosteum, from which other exostoses might develop.

There is nothing in the bursa to lead one to consider it tuberculous in origin. There are no rice bodies, no caseous foci, and no deposits on the endothelial surface resembling tubercles. I very much feared the possibility of tuberculosis here when I first saw the case, and I am correspondingly relieved to find that it is presumably of metastatic origin. We shall preserve this gross specimen for our collection when we have excised it. These peg-like exostoses I shall chip off with the chisel, together with the underlying bone, but I think I can approach them better by adding an external longitudinal incision to my internal, and can also thus better insure the complete extirpation of the sac.

[Longitudinal external incision along the outer border of the Achilles tendon corresponding to the incision along the inner border.]

Note how easy it is now to chip off the entire exostosis-bearing area of the calcaneus. Both the scars from these incisions lie in a position where they are well protected from subsequent pressure.

[Dr. Murphy completed the excision of the bursa, carefully following up each niche and corner of it in order to remove the sac *in toto*. There was no subsequent interposition of fat for fear of another bursa developing. There was very little hemorrhage. The wound was closed with deep catgut sutures and superficial sutures of horsehair and silkworm-gut. The entire operation was carried out without hand-contacting with the tissues or with any instrument at the points where it touched the tissues. Thus a sterile wound was insured, which will heal by primary intention.—Ed.]

One can readily see how a shoe with a low heel would cause much more pressure on such a bursa than a shoe with a high heel. I know from personal experience that such is the fact. Every time I cross the ocean I get a painful heel myself. When I go aboard I usually put on rubber-soled shoes without heels, always hoping against the inevitable; and after the first or second morning both heels usually become sore. Then I change back to my heeled shoes, and the pain is relieved at once, exactly in consonance with the mechanics of the problem.

The plantar bursa has been bothering this patient some, so I shall remove it also. I am making my incision along the outer side of the foot, just above the sole, to keep it out of the pressure area. See the thickened periosteum over the calcaneal spur which I have now cut down on! Here is the infected bursa lying in front of it. The greatest plantar sensitiveness was located just in front of the tubercle, corresponding with the location of this bursa. That, I believe, is the rule rather than the exception. It is the bursitis that causes the pain and disability and not the spur itself. I think this is the opinion of most surgeons and pathologists who have investigated this condition from Plettner down.

Blencke, who made an extensive investigation of normal feet with this point in view, using the *x*-ray to determine the presence or absence of the plantar spur, found the spur present in 2.8 per cent. of all apparently normal feet. His investigations covered over 600 normal cases.

My friend Partsch, the Breslau surgeon, has such a calcaneal spur himself, He was unaware of it until, in a turning exercise in a gymnasium, he missed the mattress and struck his foot on the floor. Fearing he had done himself injury, he had an *x*-ray picture taken, and the presence of the plantar calcaneal spur was revealed. The symptoms of the trifling injury rapidly disappeared without the presence of the spur having any apparent effect upon them, and he has had no further trouble of any kind from that source.

We shall next carry out the same procedure on the bursæ of the right foot. The patient has not had so much trouble with his right foot as with his left, but the plantar bursa on this side has given him more trouble than the Achilles bursa. I am making a lateral incision at the outer border of the right Achilles tendon. As I expose the bursa one can see that it is not half the size of the one we excised on the left side. As I open it a little clear fluid runs out, with the same oil globules floating on top of it. There are no rice bodies in the cavity, and there are none of those cartilaginous exostoses on the posterior surface of the calcaneus which we found on the left side. There are one or two little tags of fat in the bursa, and these I shall excise with the bursa, which is so small that the excision can readily be done, I think, from this one lateral cut. I shall not remove any of the periosteum or bone here, because it shows no evident changes, either on inspection and palpation or in the *x*-ray plate.

I am making a lateral incision again just above the sole of the foot to reach the sensitive right plantar bursa. There appears to be some little inflammatory reaction, but no bony spur, such as was present on the left side. There is also a small fatty body in this pocket, and I am excising it with the bursa. Since it lies inside the bursa, it may be and probably is a factor in keeping up the irritation of the endothelium. Note that I am excising this entire sac in a mass, and that the small body of fat is coming with it.

Visiting Doctor: What is the condition of the bone beneath?

Dr. Murphy: It is practically normal. The periosteum is somewhat thickened, but the bone itself appears not materially changed. There is no distinct evidence of osteoporosis or eburnation, and no evidence of tuberculosis, either in this or any of the three other bursæ which we have excised.

I am doing all this work without hand-contacting. It always seems a little awkward to work solely with instruments after one has been accustomed to doing abdominal work, where so much has to be done with the hand. This new technic, however, is only a matter of practice.

Visiting Doctor: Are these pains in the heel always due to a bursitis?

Dr. Murphy: By no means. The causation of pain in the heel is still an open question. It cannot always be decided. Probably bursitis is the most frequent cause; but periostitis and osteomyelitis are no doubt occasional etiologic factors. The older authors considered that these calcaneal spurs, by pressing directly on the soft tissues, with their contained nerves, produced the pain; but modern surgical opinion tends more to lay stress on the accompanying bursitis and less on the spur itself.

There is a chronic periostitis of the calcaneus which the older writers called "gout of the heel." There is a peculiar and interesting disease of the plantar fascia, a nodular fasciitis, which, like the bursitis, is probably metastatic in origin, and may produce similar severe and disabling plantar pain. A similar inflammation of the connective tissue also occurs sometimes in the Achilles tendon, a nodular tendinitis, which, however, in that location is presumed to be usually traumatic in origin, the trauma of ill-fitting shoe-tops, or actual blows or tenotomies being the more frequent causative factors.

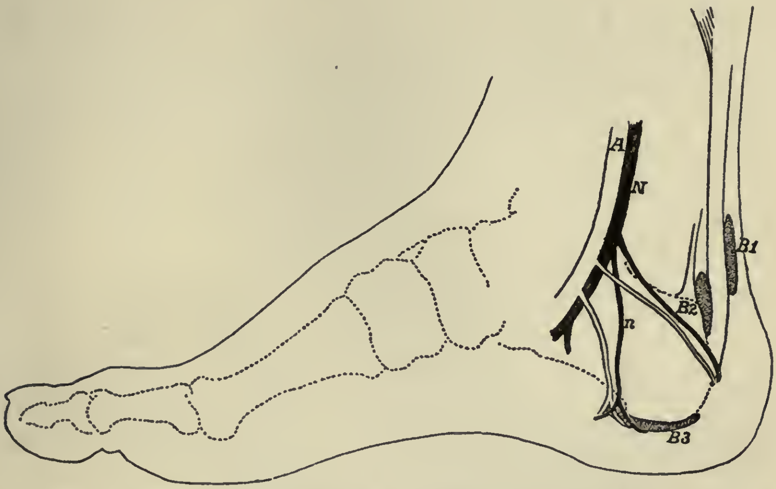


Fig. 146.—Showing the relation of the posterior plantar cutaneous branch of the posterior tibial nerve to the subcalcaneal bursa (B. subcutanea calcanea). This anatomic relation explains the intense heel pain caused by subcalcaneal bursitis at all stages of the disease. Abbreviations: A = Posterior tibial artery; B_1 = superficial Achilles bursa; B_2 = deep Achilles bursa; B_3 = subcalcaneal bursa; N = posterior tibial nerve; n = posterior plantar cutaneous branch.

The inflammations in the bursæ about the heel are particularly apt to be metastatic from gonorrhea, and the French have gone so far as to call these bursitides the "gonorrheal foot" (*pied gonorrhoique*). We are beginning to find that bursitis, like arthritis and osteomyelitis, is frequently metastatic in origin from acute and chronic focal infections, such as pharyngitis, tonsillitis, furunculosis, and the like.

Frohse, the anatomist, has made an interesting observation in connection with this plantar bursitis. He has shown by a number of careful dissections that the plantar cutaneous branch of the posterior tibial nerve frequently sends a twig directly across the infracalcaneal bursa, where it will be immediately exposed to the action of inflammatory products in the bursa as well as to pressure from the bursa when the latter is enlarged.

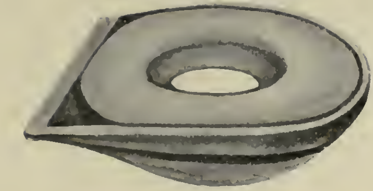
Of course, a tumor of the foot, if it contains numerous nerve-filaments or presses on neighboring nerve-endings, may also produce pain, and in the sole of the foot, besides osteomata and chondromata, we have lipomata, endotheliomata, neuromata, and sarcomata. The chalky deposits in gout occasionally come to lie between the plantar fascia and the bone, and may thus give rise to pain, as Duckworth first described. These gouty deposits give a characteristic appearance on the *x*-ray plate.

Visiting Doctor: Do these inflamed bursæ always need to be excised?

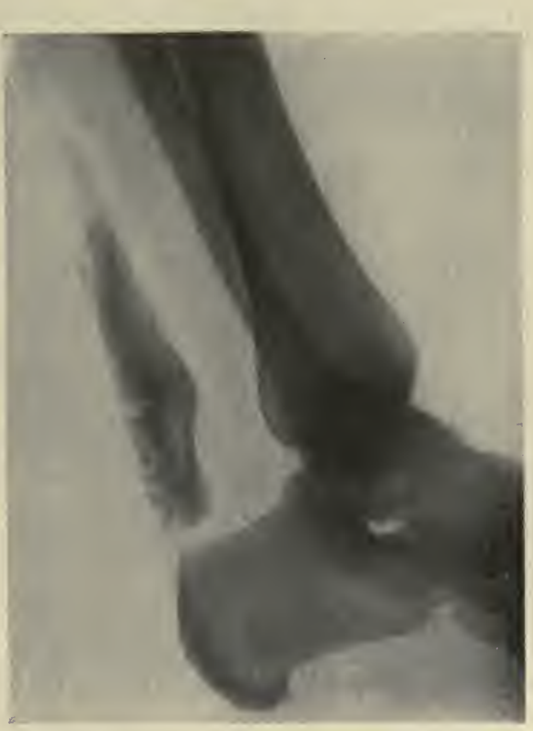
Dr. Murphy: As a rule, the patient does not consent to excision until he has run the whole gamut of non-operative procedures. There are a number of these latter palliative aids, and in mild cases they may often suffice. For the severe chronic cases, however, operation must be performed, and radically performed, to obtain a permanent cure. You can readily understand how perfectly useless it would have been to attempt to rid this patient of the pain in his left heel without excising those cartilaginous pegs which kept continually aggravating his trouble by their mechanical action.

The patients often use corn plasters, felt rings, rings of rubber sponge, and perforated felt soles. An ingenious air ring has been devised by Sarrazin to fit into a prepared hollow in the heel of the shoe and give the patient pneumatic protection for his inflamed bursa. Alcohol injections, phenol injections, and iodin injections into the bursa have been recommended after first emptying the bursa by aspiration; but this injection treatment is both painful and uncertain, and increases the difficulty of operation, when it subsequently becomes unavoidable, as it so frequently does.

The excision operation is easily performed, and, when the



Sarrazin's air cushion.



Calcification of the Achilles tendon due to tenotomy forty years previously (a half-tone from an x-ray plate, Professor Meyer's case).

diagnosis is correct, that is, when the pain is due to a bursitis, produces a radical, permanent cure, and the patients who have been incapacitated for work and suffering with pain for years are usually extremely grateful. *Correct diagnosis is the one essential.* It is no use removing the bursa if back of the bursitis lies a periostitis which is not attacked, or an osteitis, or if the trouble lies in one of those chronic nodular inflammations of the fascia rather than of the bursa.

Visiting Doctor: Is this what is known as Morton's disease?

Dr. Murphy: No. Morton's disease is metatarsalgia, not talalgia, which is the correct name for pain in the heel. In Morton's disease the pain lies much further forward in the foot, particularly over the heads of the metatarsals, and is due to an arthritis or an osteitis in this location, as a rule, though there are some cases of Morton's disease which show no changes in the *x*-ray plate.

I have had an outline of the subject of exostosis of the os calcis placed on the board, and below it I am having written in now the causes of painful heel. I find these short outlines of great help in teaching my students. They are able to remember such a bare skeleton of facts fairly readily, and in the course of the year's teaching gradually group their further information on the subject about this outline. I think that many surgeons, too, will find such outlines of use to them in analyzing the etiology and planning the management of such cases.

EXOSTOSIS OF THE OS CALCIS

Varieties: 1. Plantar. 2. Sub-Achilles.

Synonyms: *Kalkaneusspornen* (Plettner); *talalgie* (Duplay); *kalkaniodynia* and *achillodynia* (Albert).

Plettner's able paper (1900) is the basis of the literature.

Age: From fifteen to seventy-eight years.

Frequency: 2.8 per cent. to 9 per cent. of all normal persons past puberty (varying according to different statistics). Frequently the disease is bilateral.

Etiology: Unknown. It seems to bear no relation to general rheumatoid conditions. Flat-foot is an etiologic factor.

The supposed causes are: Chronic traumatic irritation; "osteoarthritic constitution"; metastatic infections, especially gonorrhea; development of sensitive and inflamed bursæ. The exostoses may be on one plantar surface, or on both. Much less frequently beneath the Achilles tendon. Low-heeled shoes a positive contributing factor (Murphy).

SYMPTOMS: Pain on pressure, lateral and direct; pain in walking, as tension is put on the tendo Achillis or on the plantar fascia.

X-ray plate confirms the diagnosis if exostosis is bony. The plate does not show purely cartilaginous exostoses.

PROGNOSIS AND TREATMENT: Palliative, the air-pillow or rubber ring. Conservative. Operative: The excision (*a*) of the process, and (*b*) of the bursa, both absolutely essential; periosteum also, if involved. Post-operative treatment: Rubber cushion. Implantation of pedicled or free fat flap (Murphy) to cover denuded bone. Never make the incision through the plantar surface of the heel nor through the tendo Achillis to the bursa; always make the cuts laterally so they will not be subjected to pressure, undergo keloid degeneration, or develop into a painful scar.

CAUSES OF PAIN IN THE HEEL:

1. Flat-foot.
2. Tumors.
3. Inflammation of bursæ, tendons, fascia, and bone (gonorrhea, metastatic infections).
4. Traumatic affections of the soft parts and bones (fractures, subluxations).
5. Neuritis.
6. Gout.

[NOTE.—The wounds healed by complete primary union and the patient made an uneventful recovery. We have not heard from him since he left the hospital, and in such cases no news generally means good news.—ED.]

CONGENITAL PERINEAL FECAL FISTULA.—ANAL INCOMPETENCE FOLLOWING OPERATION FOR IMPERFORATE ANUS.—PLASTIC CLOSURE OF FISTULA AND REUNION OF DIVIDED LEVATOR ANI

HISTORY

The patient is a single man, aged twenty-three years. He entered the hospital December 22, 1914. His family history is negative as to tuberculosis and carcinoma. Except for his present trouble, he has never been ill, save with the usual diseases of childhood and an attack of typhoid fever in the spring of 1912. There were no complications with the typhoid fever. The patient denies venereal infection.

His present trouble dates back to the time of his birth. He was born with an imperforate anus; but there was also a fistula present which led from the rectum underneath the skin to the posterior margin of the scrotum in the median line of the perineum. At the time of birth all the fecal material which was passed came through this fistula. Two months after birth an operation was performed, and an artificial perineal anus was made at a spot about where the rectum should have opened. After the operation all the fecal matter passed through this artificial anus, but the perineal fistula did not close. Both gas and mucus have escaped periodically through the fistula up to the present time. The patient has partial control of his sphincter ani; but if he indulges in articles of diet of a laxative nature, he is unable to control the consequent loose bowel movements.

The patient also has a tiny fistula over the bridge of his nose. This fistula does not discharge, save very rarely, and has caused the patient no inconvenience at all.

Examination of the patient by Dr. Murphy on December 21, 1914: The posterior portion of the sphincter ani is intact. The artificial anus has been made in surgical consonance with the natural defect. The anterior portion of the anal circle is absent; and the fibrous band representing the obliterated rectum lies behind the present artificial anus, showing where the normal anal opening should have been. There is also an indentation of the skin at the external limit of this fibrous cord, and a descensus of the rectum above it, the two membranes, skin and mucosa, almost contacting. There is a canal lined with mucous membrane extending forward beneath the skin in the median line of the perineum to the posterior margin of the scrotum, where it opens through the raphé. The continuity of the anal sphincter is interrupted only at its anterior margin. The patient has normal urinary control.

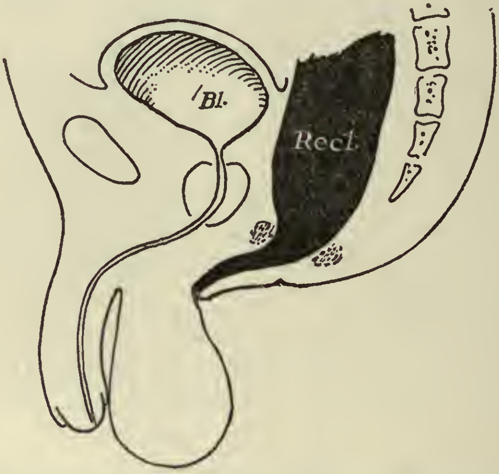


Fig. 147.—Diagram showing the type of the condition present before the first operation, which was performed at the site of the anal dimple indicated here.

In excising the perineal fistula we must be careful not to injure the urethra, and in order to protect the latter better, we must pass a sound into it before we start operating. The perineal fistula is lined with mucous membrane, which must be dissected out completely and will be retained as an apron at the anus to cover and protect the sphincter ani when it is dissected out and resutured.

COMMENTS AND OPERATION

Dr. Murphy (December 24, 1914): This is a most interesting case of embryonal defect. The patient was born with an imperforate anus. He also had a perineal fecal fistula at birth, which he still possesses, and which is one of his sources of trouble. It extends forward in the median line of the perineum beneath the raphé, and opens at the posterior border of the scrotum. Because of the existence of this fecal fistula the patient as an infant did not have an immediate and complete stoppage of the bowels. The presence of the fistula very likely saved his life. The mortality rate in infants with complete fecal obstruction due to an imperforate anus is very high, even when the condition is promptly recognized and operated. If not operated, the patients all die within the first ten days or two weeks after birth. The cases with an accompanying fecal fistula offer a much better prognosis. The vulvar or vestibular fistulæ, often wrongly called vaginal fistulæ, offer the best outlook of all the cases of imperforate anus. We had such a case earlier this year, which we reported in the Clinics (October, 1914), and which has done very well since then. After these vulvar fistula cases the perineal fistulæ offer the best chance for recovery.

This patient had his operation two months after birth. He could not possibly have survived that length of time had a fistula not been present which furnished exit for some fecal material and gas. At the previous operation an artificial anus was made just in front of the anal dimple. The external anal sphincter either was cut at its anterior margin or was undeveloped there from birth; and the surgeon failed to unite its fibers. No attempt was made at the excision of the perineal fistula. Why, I do not know. Perhaps because the patient was not in good general condition, and the surgeon aimed to do as little as possible and drain the bowel with maximum speed. Because of the interruption in the continuity of the sphincter anteriorly the patient has had no sphincter control of the bowel since the operation, and he is now twenty-three years old. You can imagine what a great inconvenience this fecal incompetence is to him.

The patient has never passed any urine through this perineal fistula. He no longer passes feces through it. There is merely a little mucoid and gaseous discharge from time to time. The connection with the rectum can be readily made out by slipping a sound along the fistula to the anal opening. There is no communication with the urinary tract. Remember that the cases of imperforate anus with a urinary fistula offer a prognosis practically as gloomy as in the cases of imperforate anus with complete obstruction. We have no reason to presume in this case that there ever has been an opening between the urinary tract and this fistula. That fact gives us a splendid prospect of securing a permanent success by this operation. On slipping the finger inside the anus one cannot feel the external sphincter anteriorly; but posteriorly one can feel the firm elastic horseshoe which the retracted muscle makes. Preliminary to the initial incision I am stuffing the anal opening with a strip of gauze, and thus prevent an escape of rectal contents. I am slitting up the perineal fistula, using a grooved director as a guide. (The accompanying illustrations show the different steps in the operation.) Now that the fistula has been opened up, you can all see the mucous membrane lining it, which runs from the anus to the very tip of the fistula. This lining we must dissect out completely. Although the fistula seemed so superficial, you can see, as we are dissecting it out, that it lay beneath Colles' fascia, because we are exposing the structures beneath this fascia, that is, the accelerator urinæ muscle and the crura penis, in our dissection. The presence of the sound is quite a help to us in avoiding injury to the urethra, as it is very essential that we should; because to make any opening in the urethra which might admit urine beneath Colles' fascia would expose the patient to the danger of an extensive urinary infiltration.

As we approach the anal margin in the dissection, we are coming close to the retracted stumps of the external anal sphincter. I am exposing them and freshening them as I free them from

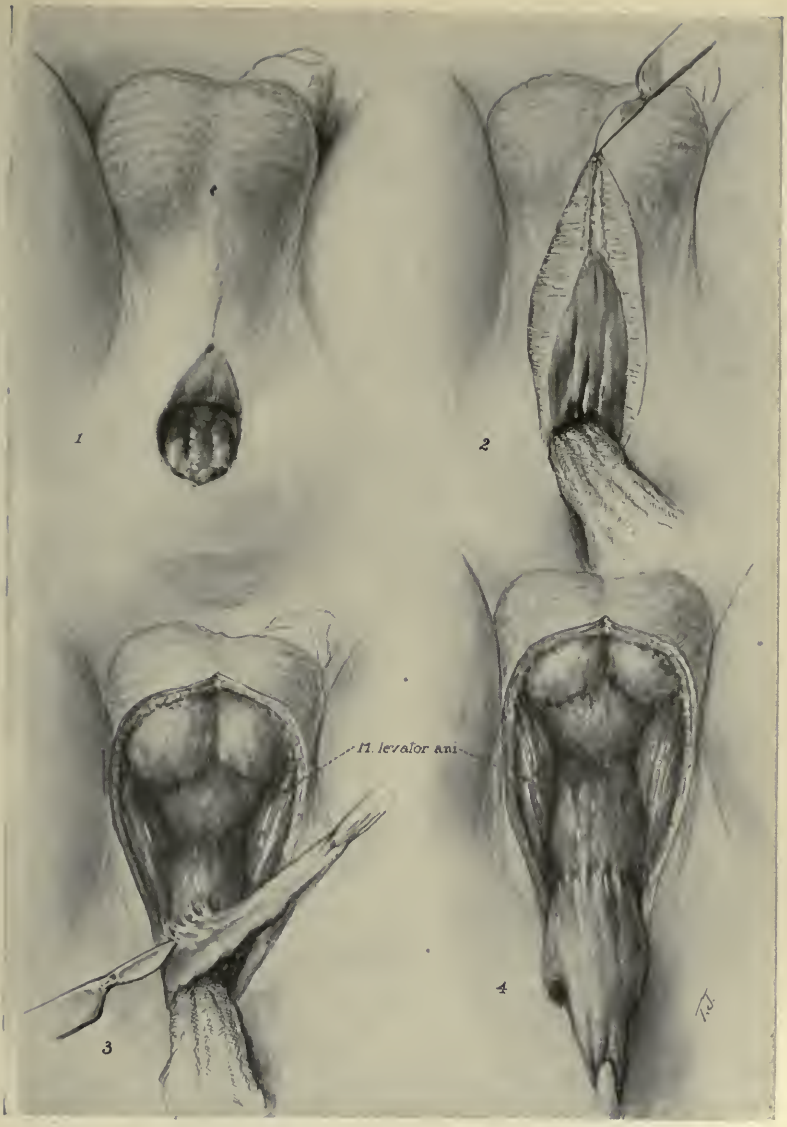


Fig. 148.—Perineal fecal fistula with anal incompetence due to congenital non-union of levator ani and sphincter ani muscles. 1, Gaping, incompetent anal opening with exuberant mucous membrane. Anal and scrotal openings of the perineal fistula shown. 2, Anal opening stuffed with a gauze strip to prevent infection of the field of operation by the escape of rectal contents. 3, Dissecting out the fistula and exposing the separated edges of the levator ani muscle, which lay very close to the skin surface at the sides of the fistula. 4, The dissection completed, the new anal opening outlined, and the exuberant mucous membrane drawn backward, showing the extent of the perineal dissection. The urethra, containing a sound and covered by the corpus spongiosum and accelerator urinæ muscle, lies at the bottom of the field. The exposed edges of the levator ani lie at either side of the wound ready for suture. The deep muscles of the perineum, including the compressor urethræ and accelerator urinæ, are intact, and, consequently, there has been no urinary incontinence.

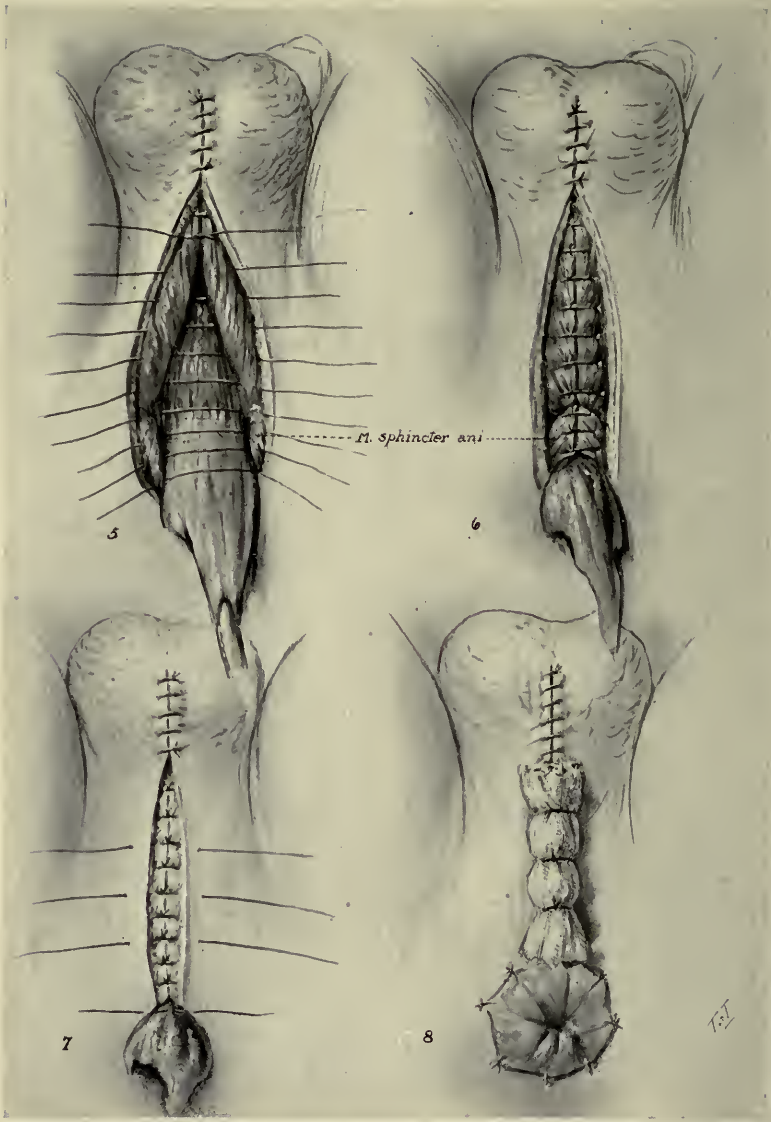


Fig. 149.—Perineal fecal fistula with anal incompetence due to congenital non-union of levator ani and sphincter ani muscles. 5, Scrotal wound sutured; interrupted catgut sutures passed through the edges of the levator ani and sphincter ani muscles. 6, Deep sutures tied, approximating the edges of the levator and sphincter ani and restoring the perineum and anal ring; exuberant mucous membrane projecting posteriorly. 7, Superficial perineal muscles and edges of Colles' fascia united by interrupted catgut sutures; deep silkworm-gut sutures (three) inserted; posterior catgut suture inserted, fastening the anal mucous membrane to the skin and preventing retraction of the former. 8, Deep sutures tied over gauze to prevent "cutting-out"; the edges of the exuberant anal mucosa sutured with catgut to the surrounding skin to prevent retraction and to protect the anal wound from fecal contamination.

this covering of mucosa. . . . The mucous membrane has now been completely freed, and you see what a large opening we must close. We shall suture the defect from its anterior margin backward, using interrupted sutures of catgut to pull together these lateral masses of muscle to meet each other in the midline. Gradually we are working back to the sphincter ani, and we are using the same interrupted deep catgut sutures to catch the exposed ends of the sphincter. See how nicely the two sphincter ends come together in closing off this tube of mucous membrane which we shall later fasten over them! The scar tissue formed here subsequently will be only on the anterior margin of the anus, and therefore there is no danger of the development of a circular scar, with subsequent atresia of the orifice. The anal opening now admits the tip of my little finger nicely, and I believe that it will be fully competent and yet elastic enough to permit an adequate passage of feces. I am sewing up the scrotal portion of the wound with a continuous suture of catgut, which will not need to be disturbed subsequently, but I am using interrupted silkworm-gut sutures to close the perineal opening, and am tying them over gauze in order to prevent their cutting through the tissues.

What shall we do now with this exuberant anal mucous membrane? We shall simply stitch it over the surrounding skin with interrupted sutures of catgut. It will thus protect the margins of the wound from infection, and we shall be certain to have enough to cover the sphincter adequately. Whatever remains unneeded, we can clip away subsequently.

VISITING DOCTOR: What were the muscle masses at the sides which you brought together in the median line?

DR. MURPHY: Those were the edges of the levator ani muscle. They had been separated for the entire distance of the fecal fistula so that there was no control of the perineal group of muscles between the anus and the scrotum. To regain this control we therefore had to suture the levator ani as well as the external sphincter. It has been an extensive operation, but we believe that we should get a good result, even if some infection should follow, which, as you see, we have taken antiseptic as well as aseptic measures to prevent. Our degree of success will be very largely a matter of the asepsis which we have been able to secure. We shall apply a dry dressing over this powder.

Visiting Doctor: Are these cases very frequent?

Dr. Murphy: The birth statistics give the frequency of the different forms of atresia ani as ranging between 1 case in 6000 and 1 case in 15,000. But most of the cases die, however, very shortly after birth, either because the complete obstruction is not recognized by the attendants or parents in time for successful operation, or because of the coexistence of other serious defects or accompanying complications; and not many of them reach the surgeon. This is only the second case of imperforate anus that we have had this year.

[Note.—The anal margin suppurated. The ends of the sphincter ani muscle retracted, but the levator ani muscles and the skin margin of the wound healed by primary intention. There is still some incontinence, though the condition is much improved over what it was previously. He will have to have another operation.—Ed.]

HYPERNEPHROMA OF THE RIGHT KIDNEY. —NEPHRECTOMY

HISTORY

THE patient is a man, aged fifty-seven years. He entered the hospital January 3, 1915. There is no history of carcinoma in his family. His mother and two brothers, aged fifty-four and fifty-eight years respectively, died of tuberculosis. His past history has no bearing on his present affection, except that he has had gonorrhea six times, with a stricture following the first infection.

His present trouble dates back to the latter part of 1911, when he first noticed colicky pain in the right loin, associated with chills and fever. He was not nauseated at this time, and did not vomit. He was not jaundiced either before or after the attack. The pain lasted for fifteen or twenty minutes and then disappeared. Before the onset of this attack he had always been able to eat all kinds of food without discomfort; but following it he seemed to lose his appetite and develop a dislike for food, although after he had taken several mouthfuls of food at a meal, he usually relished the remainder of it somewhat more.

A feeling of indefinite soreness gradually developed over his upper abdomen during the ensuing months, but no definite pain. Eating seemed to relieve the discomfort somewhat. He vomited often without being nauseated, fifteen or twenty minutes after meals, and the vomitus consisted usually of bile and the food he had just eaten. He has never vomited any blood or coffee-ground material. He would feel almost perfectly well usually after vomiting, which occurred frequently after breakfast but seldom after the other meals. Since November 20, 1914, he has belched considerable gas. This belching also seems to relieve the discomfort in his stomach. He has had no burning sensation or actual pain in the epigastrium since the initial attack. At present he cannot retain food long in his stomach. He has never passed tarry stools or visible blood per rectum.

About eighteen months ago he began passing blood in the urine. He usually had this bleeding for a day or a day and a half in a month. There was no pain associated with it. The blood was dark, stringy, and of a syrupy character, and appeared during the beginning of urination. The last portion of the urine was often quite clear, although distinctly blood-tinged in color. The patient has passed no visible blood since June, 1914, nearly seven months ago. He has lost 30 pounds in weight during the past eighteen months.

COMMENTS AND OPERATION

Dr. Murphy (January 9, 1915): What is the matter with him?

Intern: I think he has a carcinoma of the stomach.

Dr. Murphy: Why?

Intern: Because he complains of a feeling of distress after meals.

Dr. Murphy: How soon after meals?

Intern: Before he starts to eat he has a feeling of distress; but after he eats a few bites the distress often passes away.

Dr. Murphy: Yes. But if it doesn't, he vomits, and he vomits when?

Intern: From fifteen to twenty minutes after meals, but not later.

Dr. Murphy: That is correct. But when the gastric lesion is a carcinoma, the patient usually has late vomiting, because the stomach does not empty readily when the carcinoma is situated at its pyloric end, the most common seat of cancer. Remember that when the cancer is not situated at the outlet of the stomach, the stomach may empty very early and very completely because the pyloric sphincter tends to remain continually open when achylia is present. I think this fact was never fully appreciated until we began to use the *x*-ray in the routine examination of our stomach cases. The patient has also lost weight. What other symptoms has he?

Intern: He has passed blood in the urine.

Dr. Murphy: How long has he been passing blood in the urine?

Intern: For eighteen months, at irregular intervals; but he never has had a severe hemorrhage, and he has seen no blood at all in the urine for the last seven months.

Dr. Murphy: What was the character of the blood that he passed?

Intern: It was dark, stringy, and syrupy, and was seen chiefly at the beginning of urination. The entire urine was usually colored to a noticeable degree, but the first portions were syrupy or contained stringy ropes of blood.

Dr. Murphy: What about the conformation of the clots?

Intern: They were like strings.

Dr. Murphy: Were there any angle-worm clot formations in the urine? Did you ask him about that point?

Intern: No, doctor.

Dr. Murphy: That is what you should have asked. Clots which come from the kidneys sometimes show these angle-worm formations. They are not common, but they are just common enough to have their absence mentioned in the history. What were the urinary findings?

Intern: On January 4, 1915, the urine showed a specific gravity of 1014; an acid reaction; no albumin; no sugar; occasional granular casts; eight to ten white blood-cells to the field of a 1/6 objective; calcium oxalate crystals; occasional red blood-cells; many epithelial cells and amorphous urates. On January 5, 1915, the urine showed a specific gravity of 1020; alkaline reaction; no albumin; no sugar; no casts; no red blood-cells; three or four white cells to a 1/6 objective field; triple and amorphous phosphates, and a few epithelial cells.

Dr. Murphy: The patient is having no obvious hemorrhage now, and has had none lately, although there are a few red blood-cells present in his urine. He has a large tumor here in the right loin. In removing it I intend to make a subcostal incision, approach it from behind, and then dissect forward around it. The tumor is so large that I feel certain it cannot be taken out through the ordinary posterior lumbar incision, and so I am making a curved incision below the costal margin, between the latter and the iliac crest. The last dorsal nerve (the twelfth) and the ilio-hypogastric and ilio-inguinal nerves divide as they pass around forward and downward. They must be avoided in making the incision.

The course of this case is not at all uncommon for a tumor of the kidney; in fact, many tumors of the kidney have hematuria for their initial symptom. In tuberculosis of the kidney, too, long before a tuberculous ulcer develops on the mucosa of the renal pelvis, hemorrhages from the kidney may occur as the first sign of the disease. Remember that the primary focus in tuberculosis of the kidney lies most commonly in the cortex, next most frequently in the papillæ, and next in the mucous membrane lining the pelvis. Long before the granuloma ulcerates, and long before the white cells in the urine are very appreciably increased in number, hemorrhages may occur as the earliest sign of the disease. The same hemorrhagic onset is still more frequent with tumors of the kidney; and here, too, there may be first a slight increase in the number of leukocytes in the urine, and then a sudden severe hemorrhage, before the patient notes any subjective symptoms.

The very early cases of tuberculosis with initial hemorrhages can often be cured permanently without operation and without the tuberculous process ever opening into the pelvis of the kidney, and without a mixed infection ever developing. Tuberculin treatment is the chief means to this end. When tuberculosis begins in the mucous membrane lining the pelvis at the hilum of the kidney, pus and blood appear in the urine relatively early in the disease, and continue present throughout its course. Then, following this pyuria in untreated cases, a productive ureteritis subsequently develops, narrowing the lumen of the ureter. Retention of the products of infection takes place in the pelvis of the kidney, and a subsequent dilatation of the pelvis develops.

A pyonephrosis generally results then unless surgical measures are resorted to. [Incision.]

There is the external oblique muscle which I have split, thus exposing the fibers of the internal oblique. I am splitting the fibers of the latter parallel to the long axis of the muscle, so that I can extend my field as far forward as I desire—clear to the rectus border, if necessary. This is purely a muscle-splitting operation so far. Next we cut the transversalis muscle at right angles to its fibers a little below the costal margin, and enter the perirenal space. At the posterior angle of the incision I am continuing the dissection down to the true capsule of the kidney. We have thus made a wide exposure of the entire area, and can now decide what further line of procedure to adopt. We next follow the capsule forward, and outline the mass of kidney and tumor which we have to remove. . . . There it is nicely in the field! We must work around further in front of the tumor and see how we can separate it from the neighboring structures, and also note whether or not it has burst through the renal capsule at any point. . . . We must continue the splitting of the oblique muscles clear forward to the rectus border to obtain a larger opening, so that we can reach the anterior border of the tumor, which extends practically to the midline of the abdomen. Here is the intestine in front of the growth. It can be recognized through the posterior parietal peritoneum. In freeing the kidney from its present attachment to the abdominal viscera in front of it, we must keep close to its capsule during our dissection in order to avoid opening these adherent hollow viscera. I think I can bring the growth far enough forward now to place a curved clamp on the pedicle of the kidney. . . . We have the tumor now clearly in view and its vessels clamped at the renal hilum. There is the renal vein. Note the size of it. The tumor is adherent to it. Imagine yourself freeing and extirpating this immense kidney by the posterior route! There are the renal vessels resting on my finger now. Note how thick and short they are. This tumor could not have been brought out by the posterior route with safety. We are placing a double ligature on the vein. You can see the other vessels nicely now. See how well exposed they lie in the field, and what a mastery we have of the entire situation because of this well-chosen route of approach. We are putting a double ligature on the renal artery now. Dr. Nicholas Senn taught us the value of a double ligature on these large arteries.

[Dr. Murphy cut through the ligated vessels and removed the kidney with the contained tumor.]

We must next look over the field and see how much vascular oozing there is from the neighborhood. We have made a clean enucleation here. There is a little oozing from one adherent surface here, which cannot be ligated, and which we shall therefore have to cauterize. From the appearance of the surface of the growth I should judge that it is a hypernephroma. The prognosis in hypernephroma is not good. Besides making distant metastases, the growth recurs and recurs locally, but not in the opposite kidney, as does sarcoma. It has all the other characteristics of a sarcoma, however, and visceral sarcomata are very dangerous growths.

Let the record show that the peritoneum was not opened; that we exposed the tumor by a right subcostal curved incision, extending around in front clear to the border of the right rectus muscle, splitting the oblique muscles parallel to the long axis of their fibers, and dividing the transversalis muscle at its outer margin at right angles to its fibers.

Remember that a muscle divided directly transversely, if accurately reapproximated, becomes quite as strong as before it was cut. You may remember that Mikulicz, many years ago, in operating on the gall-bladder, divided the rectus muscle transversely in many cases without the slightest subsequent disability of the patient. We had a demonstration of the efficiency of muscle reunion in the case of an athlete that I have cited many times. His biceps was severed subcutaneously; it spontaneously reunited by fibrous tissue and grew as strong as ever, and he could lift more after than he was able to before the accident, so he stated. I am careful to avoid including the iliohypogastric nerve in my suture, because constricting the nerve causes pain during convalescence, and an area of anesthesia subsequently.

Let the record further show that the capsule of the kidney was exposed and the kidney enucleated; that at the upper renal pole the tumor had penetrated the capsule in one small area, and was torn in removal, so that the fragments left behind had to be subsequently taken out; that the growth was closely adherent on its under surface to the duodenum, pylorus, and to the intestines; but that the peritoneal cavity was not opened during the operation.

The prognosis in all varieties of sarcoma of the kidney is unfavorable. The best results are obtained in hypernephroma providing that the growth does not already project into the renal veins or the vena cava at the time of operation. Tumor thrombi in the veins make the outlook practically hopeless. The *x*-ray has quite a pronounced beneficial therapeutic effect in some of the hypernephroma cases, and is to be preferred to the operative procedure in some instances when the tumor is very large and has involved the neighboring structures extensively. Whether the *x*-ray will ever produce a permanent cure in hypernephroma, or whether it always ceases to have a further beneficial action after a certain initial diminution in size of the tumor has taken place is a question for the future to settle.

Note how this silkworm-gut suture is inserted. It is put in as a figure-of-8 stitch and obliterates the dead space, which would otherwise be left in the loin beneath the skin. The patient hereafter will have *x*-ray treatment regularly, beginning fifteen days from now, so that if there are any tumor-cells still remaining, their further growth will be prevented and their absorption hastened. We know that in the early stages of a malignant growth the *x*-ray sometimes has a strikingly beneficial effect. In the later stages it has practically no effect at all in most cases, and, in fact, it seems rather to stimulate the further growth of the tumor and diminish the body's resistance to it, so hastening death.

True sarcoma of the kidney offers a very unfavorable prognosis. Hypernephroma has a less unfavorable outlook, depending considerably on the time of operation and the type of the tumor; but in true sarcoma of the kidney there seems to be no prospect of cure at any time. In the splendid book written on the diseases of the kidney by Morris some years ago the author stated that he was unable to find recorded a single case of undoubted renal sarcoma that had remained well for five years after operation. These sarcomata do not tend to recur locally so much as they tend to appear in the other kidney. Hypernephroma metastases develop chiefly in the lung and bone, and these malignant emboli often have already slipped away in the blood-stream to the lung or bone before the primary tumor is excised. This patient has no demonstrable metastases now; and yet the malignant cells may be already wide-spread by this time, since it is two years since the patient had his initial renal hemorrhage.

Let us examine the tumor and see what it shows. It is a beautiful specimen of hypernephroma. [Dr. Murphy divides the tumor longitudinally through the cortex into the pelvis.] See how the tumor has filled the pelvis! Here is the kidney tissue. There is the ureter. Here is the tumor projecting free into the pelvis and almost filling it. (See the accompanying colored illustration.) We can only wonder that the patient did not have more hemorrhages and severer ones. The major portion of the hypernephroma lies on this side (the lower renal pole).

Let the record show that there were no tumor thrombi or mural metastases in the renal vessels; that they are free from the growth. That is a favorable prognostic finding.

This man's case makes an interesting story, and it is interesting chiefly because it illustrates just what occurs in the majority of such cases. If you asked me what condition causes more deaths than any other, I should say the condition of chronic procrastination or criminal delay. The moment that this man began to have renal hemorrhages was the moment that his doctor was able to diagnose a serious lesion in the urinary tract. It then became his duty first to determine which kidney the blood came from, and, second, the probable nature of the pathologic condition present. If that had been done, the patient would not be coming in here now, two years later, with the disease developed to almost its superlative degree. The nature of the tumor,

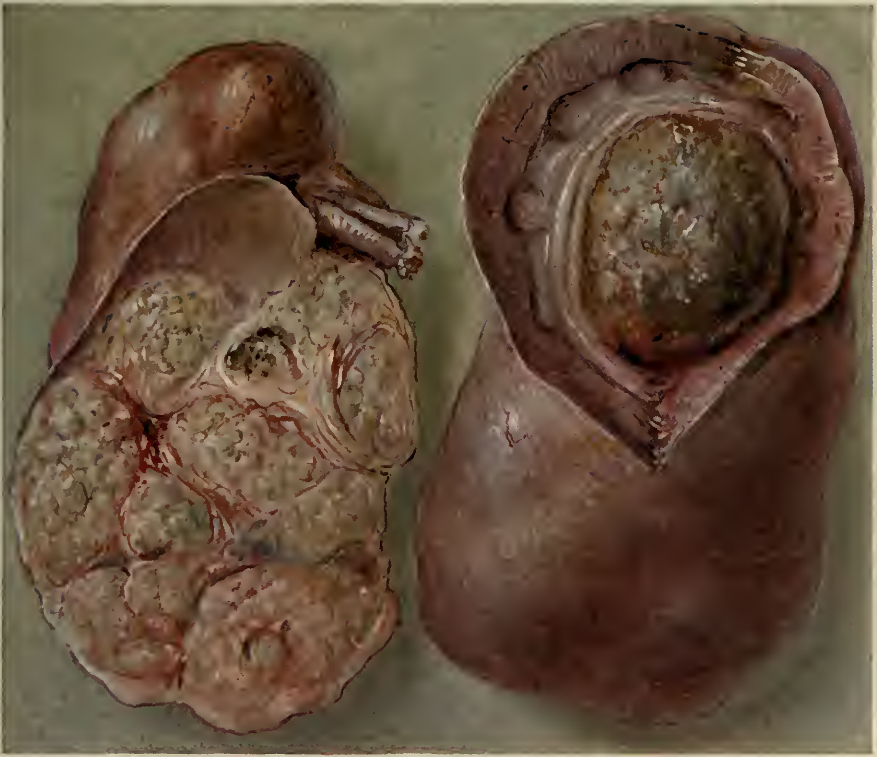


Fig. 150.—Large hypernephroma involving the lower two-thirds of the right kidney. Note the extent of the involvement and the invasion of the pelvis of the kidney by the tumor. The ulceration of the tumor in the pelvis caused hemorrhage, and the latter brought the patient to the hospital. If hypernephromata arise from adrenal "Rests," it is surprising and difficult to explain why they practically always arise in the lower two-thirds of the kidney and almost never in the upper third, which lies so much closer to the adrenal gland.

that is, its renal origin, was not recognized until the patient came here, notwithstanding its size. These errors cannot be attributed to the diagnostic incompetence of the medical profession; they are due only to its indifference and carelessness, because these tumors are often as plain as a pike-staff, and all one has to do to recognize them is to lay a hand on the patient's abdomen.

Let the record show that there were two nosophen gauze strips left in the wound and fastened with safety-pins. They will be removed at the end of twenty-four hours.

We made the diagnosis of the nature of the tumor on its gross appearance at operation, especially that peculiar orange-yellow color so characteristic of hypernephroma. We have placed on the board some statistics on tumors of the kidney.

TUMORS OF THE KIDNEY

Morris, in 154 cases, found:

Sarcomata (including hypernephroma)	in 63
(Hypernephromata, 17 per cent., G. Albarran)	
Carcinomata	in 41
Cystic degeneration	in 21
Hydatid cysts	in 11
Adenomata	in 10
Papillomata	in 3
Myxomata	in 2
Lipomata	in 2
Dermoids	in 1

Tumors of the kidney represent 2 per cent. of the total tumors of the body.

Secondary kidney tumors: Of 69 carcinomata, 10 showed renal metastases at autopsy.

Rohrer found that one-third of all the cases of malignant renal tumors occur in children. Kelynack found that 52 per cent. occur before the age of ten years, and a large proportion before the age of five years.

The mortality from nephrectomy ranged from 64½ per cent.

in the decad from 1880 to 1890, to 22 per cent. in the decad from 1900 to 1910. Individual recent statistics are Küster's (25 per cent.), Schmieden's (32 per cent.), and Israel's (20 per cent.). This mortality is far beyond what it should be if renal efficiency is accurately estimated, the cases carefully analyzed, and the route of attack adjusted to the individual case.

The percentage of permanent cures in malignant disease of the kidney has never been accurately estimated.

DR. MURPHY: Remember that when Morris wrote his book he did not differentiate between hypernephroma and sarcoma, but included all these cases under the general heading of sarcoma. Albarran later made the differentiation and showed that 17 per cent. of all tumors of the kidney are hypernephromata. Forty-one of Morris' cases belonged to the carcinoma group; so you see that carcinoma is a relatively frequent lesion of the kidney as compared with sarcoma, although not so frequent by any means as hypernephroma.

That subcostal incision which I used makes an ideal incision for this kind of a case. Remember that the ilio-inguinal and iliohypogastric nerves separate as they travel downward and forward in this region, but the eleventh and twelfth dorsal nerves run around forward higher up, thus leaving a relatively wide space through which we can open the abdominal wall without injuring any large nerve-trunk. So we were quite safe from any risk of subsequent paralysis in using this type of muscle-splitting incision here.

PATHOLOGIST'S REPORT ON THE TUMOR (DR. SWEEK): This kidney contains a large tumor infiltrating its lower pole and external border, and extending into its pelvis. The growth inside the kidney is about the size of a hen's egg, completely fills the pelvis, but is not adherent to the pelvic mucosa, so that the incision through the exsanguinated area of Hertl allows the kidney cortex to be turned outward like a capsule, releasing the tumor and revealing the calices of the kidney intact and only slightly dilated. The cortex of the kidney is much thinner than normal, and presents the appearance of the cortex of a cystic kidney or a mildly hydronephrotic kidney. The parenchyma measures from 4 mm. to 1.5 cm. in width. The entire renal mass, including the tumor, measures 4½ by 5 by 3 inches, and is irregular and rather rough in outline. On gross section the tumor is seen to be very friable, and contains a large amount of soft yellowish necrotic material. Microscopically, the tumor is a typical hypernephroma, with extensive necroses, only about one-fourth of its tissue remaining intact, and the nuclei taking the hematoxylin stain. The remaining tumor tissue is completely necrotic, and its individual cells cannot be identified.

Visiting Doctor: Do you classify hypernephroma as a sarcoma or a carcinoma?

Dr. Murphy: I classify it as a malignant tumor! I do not care whether it is a sarcoma or a carcinoma, a mesothelioma or a displaced adrenal *Rest!* I know that it is a malignant tumor, and that the kidney containing it should be excised as soon as the presence of the tumor is recognized. Let the pathologists dispute over its microscopic character and its embryologic origin. It is of infinitely more importance to us and to the public to be able to recognize these tumors early and remove them early than it is for us to indulge in fine-spun theories as to their genesis.

It is interesting to note in this connection that we found casts in this patient's urine. Stoerck, of Vienna, you know, believes that hypernephromata arise in the course of a chronic nephritis; that when a group of urinary tubules is destroyed, regeneration takes place from the remaining tubule stumps, and overregeneration—a well-known biologic phenomenon, seen under many different and curious conditions—sometimes occurs with adenoma formation; and from these adenomata, hypernephromata (or carcinomata, as Stoerck calls them) may develop. Stoerck's theory has obtained a great following, because of the large number of accurately observed cases on which it is based, and the great scientific reputation and enormous pathologic experience of Professor Stoerck. And yet many experienced pathologists, including some of Stoerck's own coworkers in Weichselbaum's laboratory, still cling to Grawitz's original view that these tumors start from misplaced adrenal *Rests*. I myself have seen a specimen of a large hypernephroma at the *upper* pole of the kidney,—most of them occur in the lower pole of the kidney, as in the case of to-day,—where the adrenal on the same side was entirely missing. The presumption was very attractive in that case that the adrenal had been displaced into the upper pole of the kidney by some embryonic perversity, and had furnished the starting-point of the hypernephroma.

Adami thinks that these tumors are of renal origin (see Adami: Principles of Pathology, vol. i, page 745 and following), but believes that they are derived from proliferating islets of nephrogenic tissue (mesoblast) derived from fetal Wolffian ducts which have never become connected with the renal tubules. Therefore Adami calls these tumors mesotheliomata instead of hypernephromata.

However, these pathologic niceties are quite aside from the main problem which confronts us surgeons; and our plain and urgent duty is to recognize these tumors earlier than we do now, and when we have recognized them, to insist on immediate operation. We must educate the public to know that hemorrhage from the kidneys is always a very dangerous symptom and demands immediate investigation and therapy.

[Note.—Patient had primary union, made an uneventful recovery, and has remained free from manifestations of the disease since operation.—Ed.]

MYELOID SARCOMA OF THE LEFT MALAR BONE.—RESECTION OF MALAR WITH THE ZYGOMA AND THE OUTER WALL OF THE ORBIT

HISTORY

The patient is a man aged twenty years. He entered the hospital April 5, 1915. There is no history of tuberculosis or malignant tumors in his immediate family. His father and mother are living and well. He has six brothers and three sisters living and well. He has never been ill before except as a child, and denies venereal infection.

He ascribes his present trouble to an accident which took place on October 20, 1914. While pitching hay in a loft he bumped his left malar bone against a portion of protruding roof. The point struck was situated about on a level with the external canthus of the eye. The injury caused him a little pain, but not enough to pay any particular attention to it, and it soon passed away. Three days later he noticed a small swelling, not quite as large as an olive, where the bone had been struck. It was not tender and he could not budge it as he felt of it with his finger, because it seemed to be attached firmly to the malar bone beneath. This swelling has slowly grown larger until it has reached its present size. It has caused him no discomfort except for an occasional slight ache. It has not been sensitive to pressure at any time. He has had no chills or fever. The swelling has always been woody hard in consistence, and his sight has not been impaired in any way before or since the swelling appeared. He has lost no weight, and feels quite well otherwise. He has applied liniments to the swelling from time to time ever since it started, but with no beneficial effect.

Examination by Dr. Murphy, April 5, 1915: The mass is woody hard. There is no bruit, and no fluctuation in it. In its removal we must excise a portion of the malar process of the superior maxillary bone, a portion of the malar process of the temporal bone, and most of the outer wall of the orbit, as well as the zygoma. It will be necessary to transplant a piece of bone from the tibia later to fill in the defect thus produced.

COMMENTS AND OPERATION

Dr. Murphy (April 7, 1915): The *x*-ray plates are seen there in the shadow-box. They do not help very much in the diagnosis of the case. Inspection and palpation really tell one more. All that one sees in the lateral *x*-ray plate is a slightly increased shadow in the region of the malar bone. The other picture, the one taken from behind forward, that is, with the face resting on the plate, shows a distinctly larger shadow over the left malar than over the right malar bone. In this shadow there seem to be two or three lighter areas of porosity, such as one sees in a myeloid sarcoma or in a chondrosarcoma. Even in the *x*-ray plate (the frontal picture) the elevation of the soft parts produced by the malar tumor is quite noticeable.

What kind of a tumor is this, is the question which we naturally ask ourselves. It is not easy to determine. The examination findings suggest a myeloid sarcoma very strongly, but the history is not quite typical for a central sarcoma, and I am always strongly inclined to give credence to a patient's statements when he insists he is right, as in this case. The patient states that no particular swelling appeared over the malar bone on the day of the injury: that it was only after three days had elapsed that the tumor appeared. The increase in size since then has been gradual and continuous, rather slow at first, but much more rapid recently.

After an injury produced by a sharp blow like this the formation of a hematoma would be one of the most likely causes of a suddenly developing swelling. But a hematoma develops usually within an hour or an hour and a half after such an accident, and then remains stationary in size until absorption begins, and then it gradually diminishes in size until it entirely disappears, except

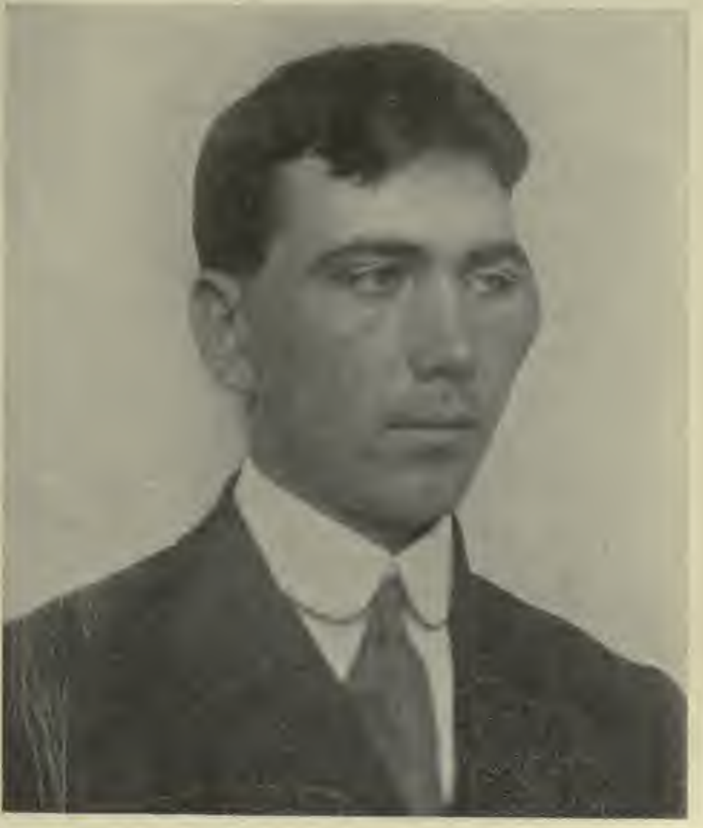


Fig. 151.—Sarcoma of the left malar bone. Photograph before operation.

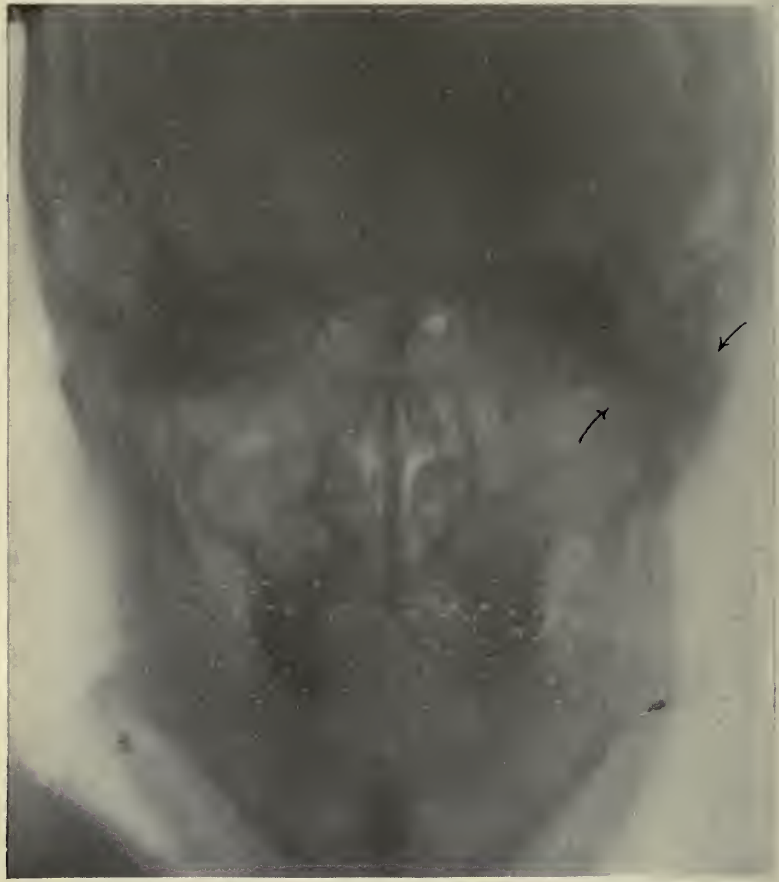


Fig. 152.—Sarcoma of the left malar bone. Postero-anterior roentgenogram before operation. Note the protrusion of the soft parts over the malar bone (better seen in the photograph) and the increased density of the left malar shadow.

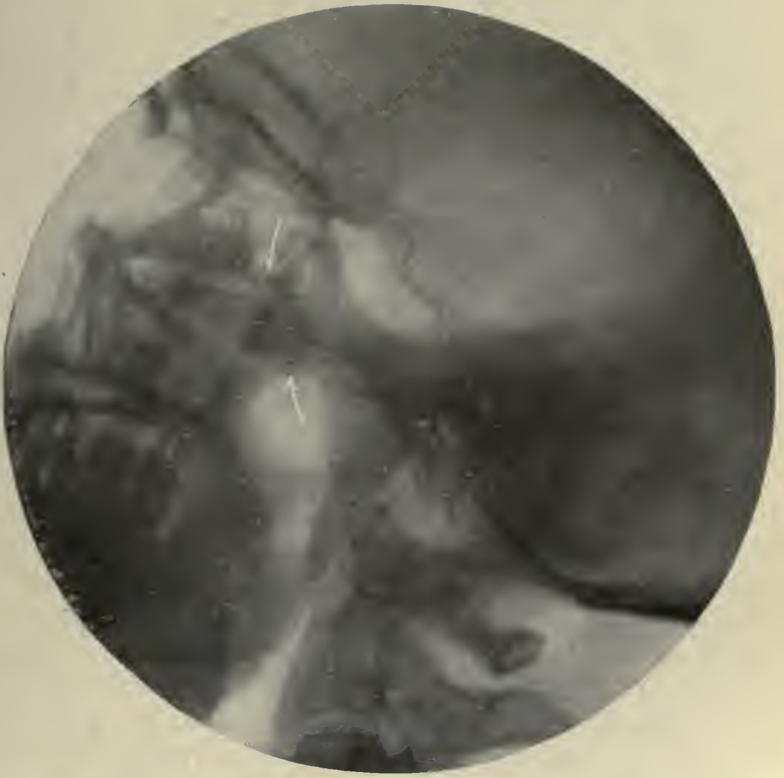


Fig. 153.—Sarcoma of the left malar bone. Lateral roentgenogram made with the left side of the head and tumor pressed against the plate (dextrosinister). Note the malar shadow somewhat increased in breadth.

in the occasional cases where a cicatricial or cystic remnant persists. The initial enlargement in this patient's case did not occur until three days after the injury. If this history is correct, it would seem to rule out the possibility of a hematoma quite certainly. So one must think of other possibilities.

There is no large vessel in this location which could suddenly give rise to a traumatic aneurysm. Furthermore, this tumor does not pulsate, nor can it be compressed; it is woody hard in consistence, not fluctuating. The blow was not sufficiently violent to knock the patient down, and therefore it was not violent enough to fracture the zygoma or the temporal bone beneath the zygoma. Therefore, the temporal muscle could not have been injured, or the periosteum beneath the muscle pulled loose, thus leading to the production of a temporal traumatic ossifying myositis. After similar blunt injuries in the region of the elbow or on the flexor surface of the arm we occasionally see the rapid development of a traumatic ossifying myositis, which is most frequently located in the brachialis anticus muscle. Such a traumatic ossifying myositis is usually preceded by hematoma formation, and the soft, fluctuating tumor thus produced often turns into a hard, firm, bony tumor in the course of a few days, as the osteoblasts from the periosteum wander through it and organize it into bone. An ossifying myositis of the temporal muscle is, however, an extreme rarity, and could not have developed from trauma here because the injury was not sufficiently violent to produce a fracture of the zygoma, without which injury of the temporal muscle would have been practically impossible. Furthermore, the patient was struck over the zygoma, and not anywhere else on the head, as he bumped against the overhanging rafter.

Might this swelling be a granuloma and not a true tumor at all? We have made the luetin test and it is negative; the Koch old tuberculin test is also negative. A syphilitic osteoperiostitis could give such a history. A syphilitic granuloma sometimes develops very rapidly after a mild trauma, and may attain considerable size in a few days. A tuberculous ostitis or periostitis practically never develops, however, in so short a period of time as three days. A tuberculous swelling very rarely, if ever, attains an appreciable size within three weeks after the initial trauma. We have no reason to consider seriously any of the other granulomata.

Might it be a bacterial infection, a periostitis, or an osteomyelitis? We have found nothing in the history or in the examination in favor of this view. The patient has had no elevation of temperature at any time and no particular local sensitiveness. There has been no fluctuation noticed at any time, and no variation in size of the tumor, which has remained from the first woody hard in consistence. We have no possibility left, then, in our differential diagnosis, except to consider this a tumor of some sort. An osteoma, a chondroma, an osteosarcoma, or a chondrosarcoma could not grow to this size in three days after the initial trauma. It might, however, have been primarily a hematoma which subsequently became malignant; a sarcoma could have attained this size in three weeks. We have no evidence that a tumor had previously originated behind the zygoma or inside the orbit, and had its rate of growth hastened by the injury. There is no displacement of the eyeball, no interference with the orbital circulation or with the movements of the eyes. There has been no disturbance of vision at any time, and there have been no headaches—in fact, nothing to indicate a lesion of the orbit which has grown outward toward the zygoma. There is also no history of a previous injury from which a sarcoma might have started, and the growth of which might have been hastened by the second injury. We must come to the conclusion in this case, I think, that the physical findings and the *x*-ray examination are of more importance than the patient's history, and that what we have to deal with is a myeloid sarcoma of the malar bone.

We shall, therefore, proceed on the supposition that this neoplasm is a sarcoma and shall make the wide excision of the tumor which that diagnosis necessitates, in spite of the great rarity of a sarcoma in this location. In carrying out this wide excision we shall run the risk of becoming involved in serious complications. First, we shall approach the parotid gland very closely, and if we do not exercise great care, we may damage the gland and have a salivary fistula develop, with the accompanying dangers of a phlegmonous inflammation in which the orbit might become involved. Second, Steno's duct may be wounded. If it is, the patient will *certainly* have a salivary fistula. Third, we must remove the outer and upper anterior portion of the orbital plate, the malar articulation of the temporal bone, and the malar articulation of the superior maxilla, besides the malar articulation of the zygoma. We must excise all the tumor, whether or not it has a sharply defined capsule. In doing so we shall not only pass over Steno's duct, but will probably also interfere with the nerve-fibers of the seventh nerve running to the orbicularis palpebrarum muscle, so that difficulty in closing the eye may persist after the operation. All these risks must be weighed in the balance against the possibility of saving this young man's life.

The large bony defect which will ensue we shall be able to fill in in a fairly satisfactory manner with a graft from the tibia if primary healing and no subsequent local tumor recurrence take place. After the removal of the tumor we shall subject the patient to a long course of treatment with large doses of Coley's serum and the *x*-ray. There may be some difficulty experienced in protecting the eyeball properly and yet exposing the entire field of operation to the *x*-ray treatment. Coley's serum, as a rule, seems to have done its best work where it was possible to remove the original tumor; but there are some exceptions to that generalization. We have a case of this kind in the hospital at present—an enormous inoperable sarcoma of the body of the ilium. It must have measured five or six inches in diameter when we first saw it, and thoroughly infiltrated this region. The patient was a druggist, a very intelligent fellow, and he readily consented to the line of treatment which we suggested. We put him on a mixed therapy, consisting of Coley's serum, the *x*-ray in large doses, and sodium cacodylate. Under the influence of this combination the mass has gone down and down in size until now it is reduced to a scarcely perceptible nodule. Whether this nodule will finally completely disappear and the patient achieve a complete cure we cannot say. I am almost tempted to try to excise it now that the tumor has become so small. Certainly the prospects of excision are not now so completely hopeless as they were when we first started our combined treatment. We know that these large sarcomata after the treatment with serum, with the *x*-ray, or radium, or even with injections of sodium cacodylate, may diminish very materially in size and may not start to grow again for a long time. But when they do start in afresh they spread much more rapidly than they did at first. Such a result is naturally very disappointing, because the patient's hopes are raised so high, as the tumor gradually diminishes in size, that he and his friends come to believe that a cure will result. Their disappointment is all the more crushing when finally the renewed and very rapid growth starts in and dashes all hope to the ground.

Gaylord and French interpret this final rapid spread of the tumor to the supercharging of the system with radium ions or with the *x*-ray to such an extent that the natural immunity of the body to sarcoma—rather slight though it may be—is still further diminished by the destructive action of the excess of radium ions, and consequently the body's resistance to the spread of the growth breaks down very rapidly. At any rate, the clinical fact is already well established that as soon as a malignant tumor, after its early retrogression under therapy, begins to grow again, its spread is very rapid and its metastases numerous and soon fatal. Whether Gaylord's explanation is the correct one or not is perhaps impossible to state at the present time; but the well-established clinical facts are exactly in consonance with his theory. Certainly the furore which radium and *x*-ray treatment at first created is gradually quieting down and the earlier hysteria in both medical and lay circles is gradually giving way to a rational plan of use of these undoubted aids in our cancer therapy.

We are making the skin incision here nearly parallel with the zygoma and a little below the center of the tumor. We next undermine the edges of the incision and retract them, thus exposing the surface of the tumor. A tumor it evidently is, with a bony shell on its outside. As I make my exploratory incision into it I find it full of little spicules of bone. We evidently have to deal with a myeloid sarcoma—a tumor of rather slow growth. In this case the history was, as you see, quite misleading. We

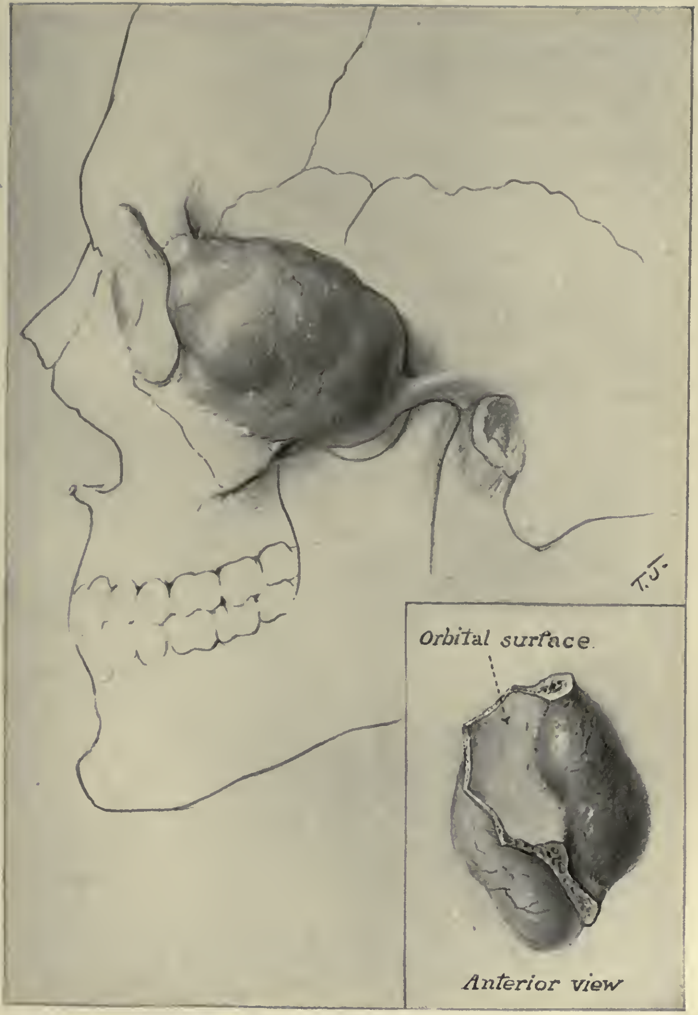


Fig. 154.—Myeloid sarcoma of the malar bone. Note the complete encapsulation of the tumor and its relation to the orbit and the skull.

do not need a microscope now to make the diagnosis. The growth is not so vascular as I thought it might be, and as I feared it might be. It seems to be fairly well encapsulated. We are freeing it from the surrounding tissues on all sides, and we are especially anxious to see whether the cells may have broken through the capsule posteriorly and invaded the orbit, or have involved the temporal bone below. If such is the case, we shall not do any more than a palliative operation, which we shall not make so extensive as we should in case we found the tumor apparently an operable one.

I am slitting up the temporal fascia, and I intend to remove it with the tumor. Now we have the posterior surface of the tumor exposed. The growth seems to have broken through the capsule here in behind and to have gone through into the temporal muscle. The temporal bone, however, does not appear to be involved. By making a wide excision of the muscle I think that we shall get all the growth. We are first cutting through the zygoma posteriorly, in order to swing the tumor forward. We next cut its articulation with the superior maxilla, so that we can lift the tumor and the malar bone away from the orbit. Lastly, we cut through the maxilla below the orbit, almost as far forward as the infra-orbital foramen. . . . The tumor now is practically free; there are only a few of the soft parts still to separate.

As I examine the tumor now that it is out I see that I was in error about its having broken through the posterior capsule. The posterior portion of the capsule seems to be intact, and so does the rest of the capsule. As I strip the temporal muscle from the tumor, I see that it comes away, leaving the capsule clean. It is probably just as well that I excised this portion of the muscle and temporal fascia, because its removal makes us doubly sure of our ultimate result, just as does the removal of the pectoral fascia in the operation for cancer of the breast. The only point of the tumor which is open to the outside is on the anterior surface, where I deliberately made an incision into the growth in order to confirm the diagnosis and immediately closed and protected it to prevent the escape of cells. I think that this patient has a fairly good chance for his life, and I feel happy that we have been able to excise the tumor completely, at least so far as we are able to estimate at present. As soon as the wound is practically healed, the patient will be given *x*-ray treatments, just as we have planned, and the Coley's serum and sodium cacodylate injections will be started at once.

The next part of our task is to diminish this patient's subsequent deformity as much as possible. I think that we shall be able to give him a very satisfactory result. I am fixing the external muscles of the globe to the temporal fascia, so as to prevent any undue traction on the eye tending to pull it inward. I have a somewhat similar case under observation at present, a young lady who was operated elsewhere and who now has a very marked and disfiguring outward displacement of the eye. We intend to transplant some bone into the defect for her, in order to remedy her facial disfigurement. We shall do the same for this patient of ours, but we shall not do it to-day, because we have had our gloved fingers in the wound, and there has been some secretion from the eye escape into the field, which means a danger of infection—and we do not wish to run the additional risk of infection which the implantation of a foreign body implies, since our field of operation lies very close to the eyeball and to the loose tissue of the orbit, in which an infection is always a very serious matter. Later, when we make the transplantation, we shall have a clean field in which to operate. We shall then take the necessary plate of bone from the front of the tibia and implant it in such a way as to take the place of the missing zygoma, malar bone, and orbital plate, and thus hold the eye plumb in place by preventing outward dislocation.

We exposed the filament of the facial nerve to the orbicularis palpebrarum muscle early in our operation. You may have noticed how I protected it from the lower side rather than from the upper, and I hope that we have not traumatized it or interfered seriously with its function. I did not mind cutting into the temporal muscle and even excising a portion of it, because that part of the procedure will have no particular influence on the patient's ultimate comfort or looks; but I *do* mind producing

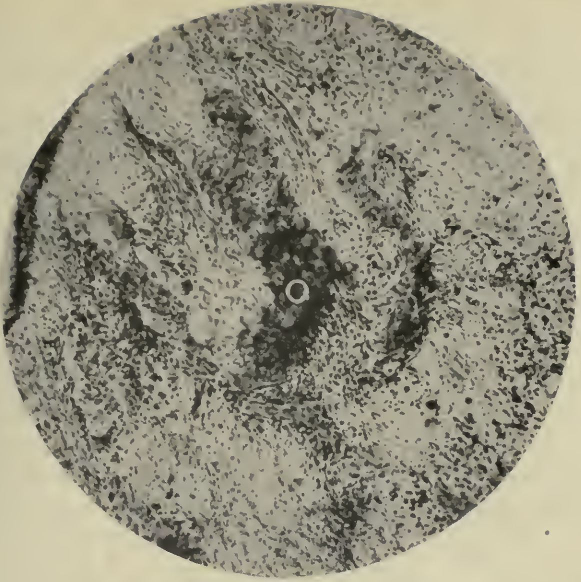


Fig. 155.—Myeloid osteosarcoma of the left malar bone. Note the matrix of spindle-cells, the occasional giant-cells, and the deeply staining area of osteoid tissue (o) in the center of the microscope field.

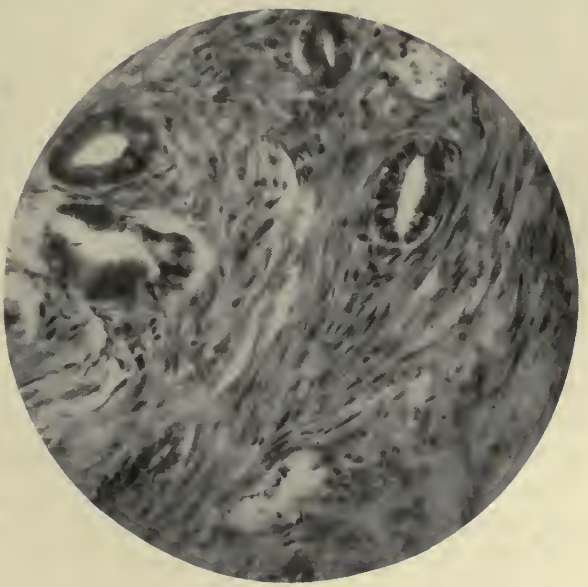


Fig. 156.—Myeloid osteosarcoma of left malar bone. In this photomicrographic field the spindle-cell matrix of the tumor predominates, and there is no appreciable calcium deposit here. This section was made from an area where the tumor was approaching the lower surface of the skin; hence the inclusion of cross-sections of the cutaneous glands.

any functional incapacity of the palpebral muscles. We are running this single stitch through the lids at the outer canthus of the left eye, in order to insure their remaining closed. The stitch will do no harm and will free the patient from any danger of subsequent lagophthalmos and corneal ulceration. We do this simply as one of our routine precautions, for I do not think that we shall have any palpebral paralysis here subsequently, because we have handled the nerve filaments to the lid muscles so carefully during the entire operation.

This variety of osteal sarcoma is one which remains a local lesion for a long period of time, as a rule; it very seldom gives rise early to any local or distant metastases. Since Peyton Rous showed a filterable virus to be the cause of an osteochondrosarcoma in the chicken, we have come to look on these myeloid sarcomata in a somewhat different light. It may be that, after all, they are parasitic in their etiology. We know what a long time it took the pathologists to demonstrate the parasitic etiology of some of our well-known granulomata; for instance, actinomycosis, syphilis, etc. It may be that we shall ultimately transfer a number of the present sarcomata to the microörganismal group of diseases. Now that Rous has shown three varieties of sarcoma in the chicken to be due to individual and distinct filterable viruses, and since Fibiger has shown that epithelioma of the esophagus and stomach can be produced in rats by feeding them cockroach nematodes, it is quite possible that we are on the brink of the discovery of other parasitic etiologic factors for a number of our present so-called malignant tumors. I have recently been collecting autopsy statistics on sarcoma and carcinoma in order to determine how long these malignant tumors remain a local disease, and when and where their metastases occur most frequently. I am not yet ready to give out the figures on sarcoma, but I have collected the statistics of 10,315 cases of carcinoma. Of these cancer patients, 19.7 per cent. had no demonstrable metastases at autopsy. It is not likely that any metastases present were overlooked because these postmortem examinations were performed by as expert pathologists as the world possesses, the prosectors at the Virchow Pathologic Institute of the Charité Hospital in Berlin. Therefore, you see that practically one-fifth of all these cancers were still purely a local disease when they destroyed the patient's life, not even the regional glands being involved.

That observation was a great surprise to me, and will be, I think, to most surgeons who peruse these figures. It points out clearly the path we must pursue if we expect to cure a greater percentage of these cases. For if one-fifth of them have no metastases even at the time of death, how much larger a percentage must have no metastases within two or three or four months, or a half year, after the primary tumor is first noticed? How much brighter that pathologic discovery makes the prospects for cure of these cases by early operation! this early operation need not always be extensive, but *it must be rational.* The primary tumor must be removed and so must the regional glands. One of the striking points in the study of these cancer statistics is that the regional glands are first in order of frequency of involvement, and the liver comes next in frequency in every form of cancer except those occurring above the level of the clavicle. Certain of the organs of the body seem to possess a very high degree of immunity to cancer-cells. The liver seems to possess a relatively low immunity; and, because metastases are so frequent in the liver, and since they appear usually to reach it by the blood-stream, we are forced to assume that the other organs of the body likewise frequently have metastatic deposits lodge in them, but do not act as so suitable a location for the growth as does the liver, perhaps because of different metabolic conditions, and so do not permit the malignant deposits to develop further in them.

[PATHOLOGIST'S REPORT: The tumor is a medullary sarcoma of the giant-cell type, with relatively few mitotic figures. It is probably only mildly malignant.]

[NOTE.—The case went on to a primary healing. There was very little deformity, but this will probably increase as the cicatricial contraction advances. The *x*-ray, Coley's serum, and sodium cacodylate will be instituted at once.—ED.]

MALIGNANT EPULIS OF THE MANDIBLE (GIANT-CELL SARCOMA).—EXCISION

HISTORY

The patient is a seven-year-old girl. Her mother brought her to the hospital January 8, 1915, because of a fleshy tumor mass in the median line of her mandible. The patient's father died of tuberculosis at the age of thirty-seven years. Her mother has had three miscarriages. The patient has had no serious previous disease.

The present trouble dates back to October, 1914, when the patient's mother first noticed a small tumor, about the size of a pea, on the anterior surface of the alveolar process of the mandible, about in the median line. This tiny tumor gave the patient no trouble whatever at the time, nor had it previously. By November 1, 1914, it had increased considerably in size and looked like a "gum-boil," so the mother states. The patient was treated locally with silver nitrate and "violet rays," and was also given a prescription to take internally. Since November 1 the tumor appears to have practically stopped growing. There is still no pain connected with it, it causes no difficulty in eating, and the patient can move her jaw freely, practically unimpeded by the tumor, which has, however, somewhat crowded her teeth inward and a little apart.

Examination by Dr. Murphy, January 7, 1915: The tumor looks like a rapidly growing malignant epulis, apparently originating from the peridontium on the anterior side of the tooth cavity, and therefore wedging the teeth backward and apart. The growth is already a little larger than half the size of an almond. The teeth, although pressed back and spread laterally, have not been much loosened. The permanent incisors were already in place when the tumor began to grow between them.

The patient has lost six weeks' time with the "violet-ray" treatment and the internal medication, which have had no apparent influence on the growth, just as one would expect. Although the superficial submaxillary lymph-glands and the glands along the facial vein on each side are already somewhat enlarged, but perfectly movable and not especially hard, the glands situated at the anterior pole of both submaxillary salivary glands do not appear much larger than normal.

A piece of the tumor removed under gas anesthesia for microscopic examination was reported by the pathologist to be a giant-cell sarcoma, with many mitotic figures, and therefore apparently rapidly growing.

Examination of the urine on January 7 was negative, except that it showed 16 leukocytes to the 1/6 objective field. No Bence-Jones bodies were found.

COMMENTS AND OPERATION

Dr. Murphy (January 9, 1915): This case is an unusually interesting one from both a diagnostic and a pathologic standpoint. The tumor has the typical appearance of a malignant epulis of the giant-cell sarcoma type. The common epulides, you know, are of two varieties, and they both spring from the same source, namely, the periosteum of the alveolar process, either outside the alveolus or inside, as here, where the periosteum is known as the peridontium. One of these common varieties is a fibroma, which consists entirely of fibrous tissue, is slow in its growth, does not metastasize, and is not at all malignant, except that it is very apt to recur locally if not completely excised. The other common variety is the giant-cell sarcoma, a case of which we have to operate to-day. The giant-cells in these neoplasms are scattered through a matrix of connective tissue and numerous round- and spindle-cells. The tumor is sometimes rapid in growth, as here, rarely metastasizes, but usually remains a local lesion, like the fibroma, confined to the alveolar process until it is meddled with surgically. Then, unlike the fibroma, but like all the mild sarcomata, when repeatedly irritated without being completely excised, it tends to recur rapidly,

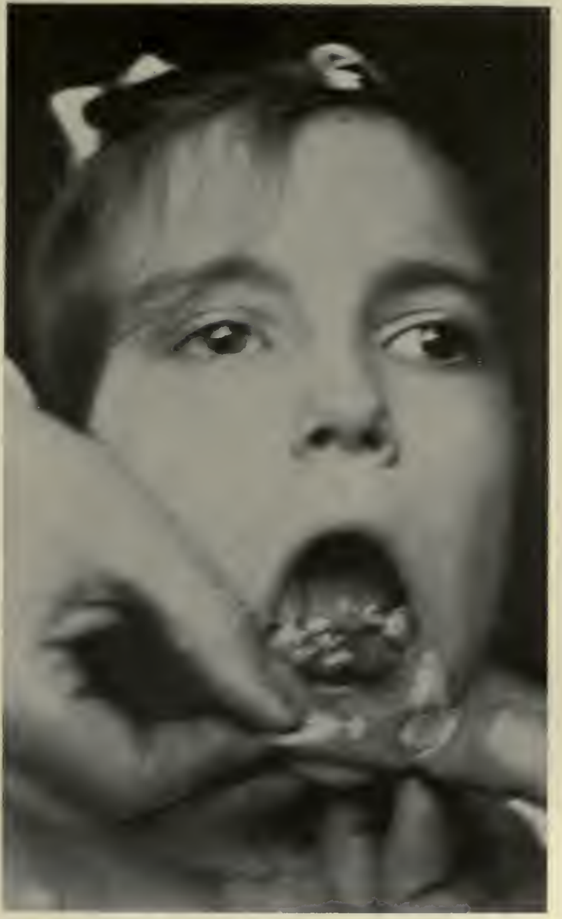


Fig. 157.—Giant-cell epulis of the mandible. Note the dark tumor springing from the alveolar margin of the lower incisor teeth and pushing the latter backward and apart.

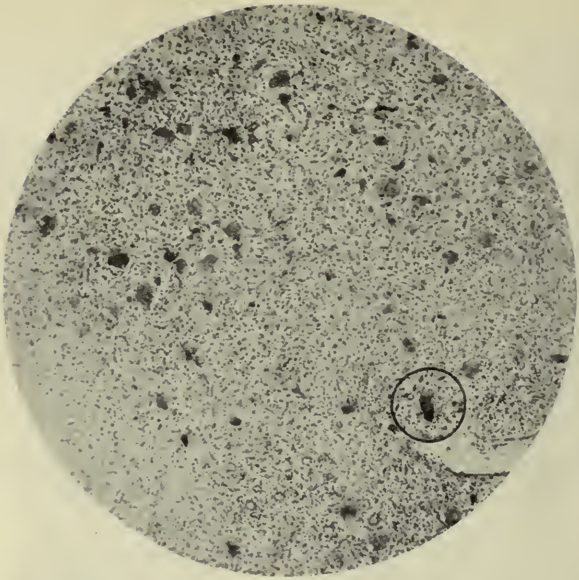


Fig. 158.—Malignant epulis of mandible (giant-cell sarcoma). Note the numerous giant-cells scattered through the spindle-cell matrix. (The circle indicates the area selected for high-power magnification.) Note the numerous small blood-vessels in the lower quadrant of the photograph. A malignant epulis is usually quite vascular, as here.

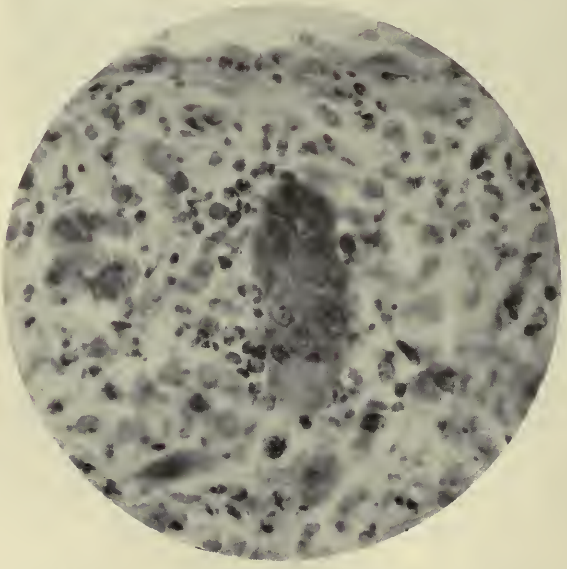


Fig. 159.—Malignant epulis of the mandible (giant-cell sarcoma). One of the giant-cells and the area immediately surrounding it (see circle on the low-power photomicrograph) has been selected for higher magnification. Note that the giant-cell is a syncytial structure containing several nuclei rather poor in chromatin. Besides the groundwork of tumor cells, there are numerous polymorphonuclear leukocytes in the field, especially around the giant-cell.

to form metastases, and, under such circumstances, may terminate fatally. It is perhaps the least malignant of all so-called malignant tumors, and has so little tendency to form distant growths that we generally regard it as a benign neoplasm. Even in the rare cases when it does cause death it brings it about through great increase in size, ulceration, necrosis, and subsequent pyogenic infection or aspiration bronchopneumonia. The growth is not at all malignant in the sense that it tends to destroy life rapidly. There are other varieties of epulis, but they are much less frequent than these two mentioned. Spindle-cell sarcoma, fibrosarcoma, and osteofibrosarcoma are sometimes seen, and also mixed tumors and tumors intermediate in character between those already enumerated. For instance, the simple fibromata sometimes contain spicules of bone, and so may the giant-cell sarcomata. All these tumors are apt to contain pigment deposits, and, because of this pigment and the numerous blood-vessels, the growths are usually dark purple in color and bleed very readily.

This is a typical case, occurring in a female patient, as is the rule, but at a much earlier age than the average, although scarcely any age seems to be exempt. A curious fact which I have occasionally noted about these tumors in adult women is that they may grow larger during pregnancy and then become smaller again after the puerperium. This observation makes me think that since the glands of internal secretion have some evident influence on their growth, they may possibly also be a factor in their etiology. At any rate, this curious and interesting phenomenon has had very little attention called to it hitherto.

Here is the neoplasm—dark, firm, not at all tender, and already destroying the local structures from which it springs. How could one continue treating such a condition long without recognizing its malignancy? We as a profession must learn to recognize these malignant conditions, and recognize them early. Delay is fatal. Early diagnosis is the only chance we have to improve our percentage of cures. There is absolutely no use in temporizing with such a dangerous condition. To delay is to throw away the opportunity of permanently curing the growth while it is still a local disease, before it has had time to metastasize to distant regions. The longer we allow such a tumor to stay in place, the more it has an opportunity to sensitize the body-cells, that is, to produce an anaphylaxis, along with which goes a marked lowering in the patient's already very limited immunity to the growth, as Gaylord has so ably pointed out.

In operating we endeavor to traumatize the tumor and the tissues surrounding it as little as possible, because, in the first place, we do not wish to squeeze any tumor-cells out into the surrounding tissue spaces, and, in the second place, because we do not wish to diminish by injury whatever local immunity to the tumor growth the adjacent tissues may possess. We shall have to begin our operation by extracting the four lower incisor teeth. We should like to be able to replant these teeth, but that will be impossible, because we must remove all the alveolar processes, together with the tumor which is springing from them. There will not be enough bone left to make sockets. We shall save the teeth, however, and the dentist will very likely be able to use them in fashioning a bridge, anchored to the adjacent teeth, which will secure fairly well in place those which we have just extracted.

We are packing gauze underneath and at the sides of the tongue, and fastening it there with forceps, so as to keep the blood from running back into the patient's throat while we work. . . . I am making my incision through the mucous membrane, somewhat outside the tumor, and am using the artist's chisel to chip away the bone and periosteum beneath the tumor, thus removing the latter together with the structures from which it springs. [Completes the removal.] Here is the specimen. I think I have removed it all. One must watch these tumors, however, for a long time after operation, to make certain that no local recurrence takes place. If there is any recurrence, it, too, must be excised as soon as seen. In such a case one is not justified, I believe, in taking out the mandible or even any considerable portion of it beyond the immediate vicinity of the growth when the microscopic diagnosis is an epulis of the giant-cell sarcoma type. As a rule, the operation which we have just performed is entirely adequate to care for the local condition.

I am going to remove the small glands beneath the patient's chin because they may just possibly be malignant, and, if malignant, must certainly be removed. Furthermore, we want to know these facts because our prognosis will be very greatly influenced by them. We want to be certain in our own minds, and be able to assure the patient and her parents, that this is a purely local growth which we have removed completely by our operation.

[Dr. Murphy then made the usual curved incision beneath the jaw for exposing the submaxillary triangle on each side.]

I am dissecting down on each side to the small glands which I felt superficially just outside each facial vein. . . . I am splitting them open now that I have them excised, in order to see if there is any gross evidence of metastases in them. There appears to be none. Next we shall remove each of those two little glands which we felt further in front. . . . Now that we have these latter in view I can see that I was in error yesterday in my diagnosis in regard to their location. They are located at the anterior pole of the submaxillary salivary gland on each side. Yesterday, as I examined them, they did not appear to be inside the gland itself, but now that they lie exposed I can see that they lie inside the capsule of the gland. . . . As I cut them open in turn they, too, show simply a lymphatic hyperplasia and no sign of malignancy. This is the rule with a giant-cell epulis. It is not infrequently accompanied by an inflammatory hyperplasia of the glands below the chin, but very seldom by tumor metastases in the glands. We shall have the pathologist confirm this diagnosis by making serial sections of the glands. This inflammatory glandular hyperplasia is the result usually of concomitant infections in the alveolar processes, the teeth, and gums. I can find no such infectious focus in the mouth of this little patient now; but children are so prone to enlargement of the cervical lymph-glands that I attribute no pathologic significance to finding them in this case.

I am sponging out our field of dissection on both sides of the neck with a solution of 5 per cent. phenol, in order to catch any stray sarcoma-cells which may be lying about, and also to diminish the chance of subsequent infection—and the latter is a far greater danger than the former, because we have been working here so close to the salivary glands. . . . Now that we have closed the submaxillary wound, I am suturing the mucous membrane of the mouth over the base of the mandibular excision. You see we have plenty of the mucous membrane left to cover in all the raw bony surface. It would not surprise me to get practically perfect primary healing here in the mouth.

VISITING DOCTOR: Just what do you mean by epulis?

DR. MURPHY: Epulis is not a very concrete term, and does not seem to mean the same thing to every surgeon. Virchow, if I remember correctly, was the originator of the term, which comes from the Greek word meaning "gums." Virchow used the expression originally to include the entire group of tumors springing from the gums; but of recent years there seems to be a marked tendency to confine its use only to the fibromata and sarcomata springing from the periosteum of the alveolar processes or their vicinity.

There are a few other rarer conditions which must occasionally be differentiated from epulis which I have not yet mentioned. One of them is the dental pulp polyp, which springs from the nerve-canal of a diseased tooth, and represents practically exuberant granulation tissue. When the crown of the tooth is broken off this pulp polyp may spread over the buried roof of the tooth, concealing it from view, and resembling closely a small epulis springing from the adjacent alveolar process. Its treatment, of course, is chiefly a matter of dentistry, by excising or pulling out the infected root. Central osteosarcoma and also fibroma of the maxillary bone sometimes occur; but they are much rarer tumors than epulis, and make much larger growths (see the accompanying illustrations), which tend to involve more or less of the entire mandible, and require very wide excision of the bone to produce a cure. Osteosarcoma of the lower jaw is not so benign as the sarcomatous form of epulis. Carcinoma arising from the mucous membrane of the alveolar process or

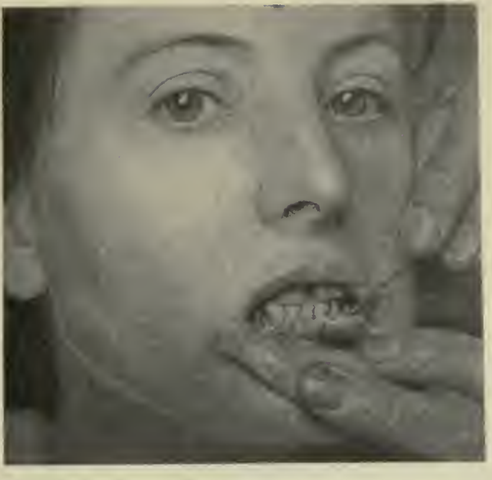


Fig. 160.—Epulis (giant-cell sarcoma) of mandible (Perthes).

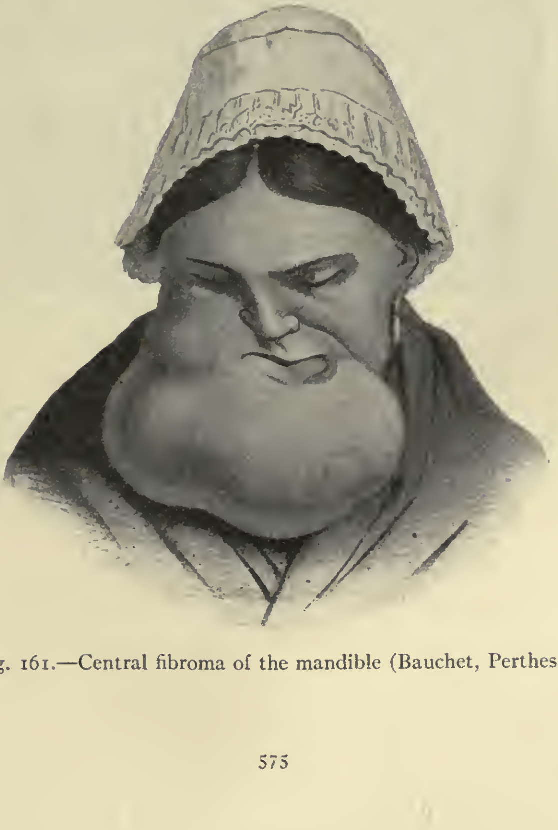


Fig. 161.—Central fibroma of the mandible (Bauchet, Perthes).

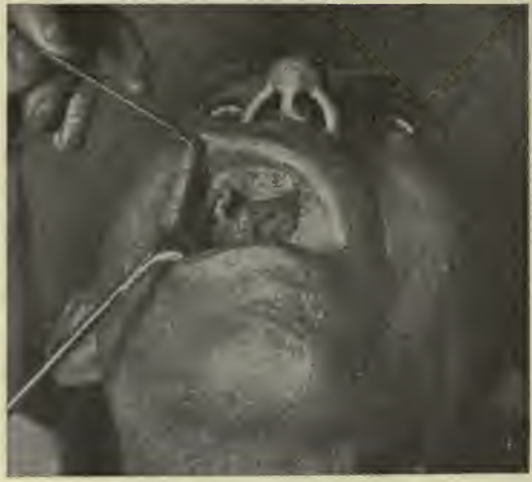


Fig. 162.—Carcinoma of the alveolar process (after Perthes).

gums is also occasionally seen. It produces an early ulcerating and bleeding growth, and is seen chiefly in the aged. It soon makes regional and distant metastases, and is rarely, if ever, cured by any method at present at our command, except very early wide excision. It is the most hopeless of all the malignant growths of the gums with which I am familiar.

Don't refuse to excise a malignant tumor of the mandible because you don't know how to fill the defect thus caused. Remember that the region of the jaws is practically the only part of the body where a foreign body will heal in place in the presence of infection. Not only will a transplant of bone heal in place here fairly readily, but even such foreign material as metal bridges and wire or metal prostheses will usually remain in place with proper measures. We have a wire frame used to supplant half of the mandible which has been in place about six years, completely supporting the remaining half of the jaw, overcoming all the external deformity, and without the slightest discomfort; notwithstanding the suppuration which primarily surrounded it, it later became and remained completely encapsulated. You get some idea of the resistance of the lower jaw to infection when you remember that practically all fractures of the mandible are compound fractures from the start and open into an infected cavity—the mouth. How rarely we see serious septic sequelæ as a consequence!

[NOTE.—Complete primary union. Patient was examined May 10. No evidence of recurrence. Serial sections of the excised submaxillary lymph-glands were examined by the pathologist and were reported to show "hyperplasia, probably inflammatory in origin, but no evidence of sarcoma metastasis."—ED.]

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Clinics of John B.Murphy at Mercy Hospital,
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